HISTOPATHOLOGICAL STUDY OF GLOMERULAR DISAESE (IN PATIENTS WITH SIGNIFICANT PROTEINURIA) – A STUDY OF 50 CASES

Dissertation Submitted to

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GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
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CERTIFICATE

This is to certify that the dissertation titled "HISTOPATHOLOGICAL STUDY OF GLOMERULAR DISEASE (IN PATIENTS WITH SIGNIFICANT PRTEINURIA) – A STUDY OF 50 CASES" **is the bonafide original work of** DR. R. CEASER **in partial fulfillment of the requirements for** M.D. Branch-I (General Medicine) **Examination of the Tamilnadu DR. M.G.R Medical University to be held in March 2007. The Period of study was from August 2004 to August 2006.**

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DECLARATION

I, **DR. R.CEASER.,** solemnly declare that dissertation titled **"HISTOPATHOLOGICAL STUDY OF GLOMERULAR DISEASE (IN PATIENTS WITH SIGNIFICANT PRTEINURIA) – A STUDY OF 50 CASES"** is a bonafide work done by me at Govt. Stanley Medical College and Hospital during August 2004 to August 2006 under the guidance and supervision of my unit chief **Prof.Dr.A.K.GEETHA DEVI M.D**, Additional Professor of Medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place : Chennai.

Date :

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INTRODUCTION

INTRODUCTION

Injury to glomeruli results in a variety of signs and symptoms of disease, including proteinuria, hematuria, azotemia, oliguria, edema and hypertension. Specific glomerular diseases tend to produce particular syndromes of renal dysfunction; although multiple glomerular diseases can produce the same syndrome. Evaluation of pathogenic features identified in a renal biopsy specimen may be required for definitive diagnosis. In patients with glomerular disease, renal biopsy provides tissue that can be used to determine the cause, predict the prognosis, and direct the treatment. In the absence of comprehensive knowledge of disease etiology, most glomerulopathies are still classified and named according to their morphologic features. The major inflammatory glomerulopathies are focal proliferative glomerulonephritis (termed mesangial proliferative glomerulonephritis if the proliferating cells are predominantly mesangial cells), diffuse proliferative glomerulonephritis, and cresentic glomerulonephritis. These diseases typically present with a nephritic- type active urine sediment characterized by the presence of red blood cells, red blood cast, leukocytes, and subnephrotic proteinuria .The severity of renal insufficiency varies in proportion to the degree of glomerular inflammation.

AIM

AIM

To evaluate the renal histopathology of patients with glomerular disease (with

Significant Proteinuria > 1 gms / 24 hours)

REVIEW OF LITERATURE

The glomerulus is a modified capillary network that derives an ultrafiltrate to Bowman's space, the most proximal portion the renal tubule. Approximately 1.6 million glomeruli are present in two mature kidneys and collectively they produce 120 to 180 L of ultrafiltrate daily. Glomerular filtration rate is dependent on glomerular blood flow, ultrafiltration pressure, and the area and composition of the filtration barrier. These parameters are tightly regulated through changes in afferent and efferent arteriolar tone and mesangial cell contractility. Arteriolar tone and mesangial cell contractility are in turn, modulated by neurohumoral factors, local myenteric reflexes, and endothelium-derived vasoactive substances. In health, the glomerular endothelium is also antithrombotic and antiadhesive for leukocytes and platelets, thereby preventing inappropriate vascular thrombosis and inflammation during the filtration process. Filtration of plasma proteins and all blood cells is normally prevented as a consequence of fenestrated glomerular endothelium, basement membrane, and foot processes and slit diaphragms of visceral epithelial cells. In keeping the physiological function of glomerulus, virtually all glomerular injury results in impairment of glomerular filtration and/or the inappropriate appearance of plasma proteins and red blood cells in the urine.

The major morphologic patterns affecting the glomerular filtration barrier of proteins, namely the glomerular basement membrane and visceral epithelial cells, are membranous glomerulopathy, minimal change disease and focal and

segmental glomerulosclerosis (FSGS). These entities typically present with nephritic – range proteinuria and the presence of relatively few red blood cells, leukocytes, or cellular casts. As a consequence of the heavy proteinuria, nephrotic syndrome was associated with hypoalbuminemia, edema, hyperlipidemia and lipiduria and a prothrombotic state. Membranoproliferative glomerulonephritis, as the name suggests, is a hybrid lesion that presents with a combination of nephrotic and nephritic features.

DETERMINANTS OF GLOMERULAR INJURY

The important determinants of the extent and severity of glomerular injury, and accordingly of the clinical presentation, include

1. The nature of the primary insult and the secondary mediator systems that it provokes
2. The site of injury within the glomerulus and
3. The speed of onset, the extent, and the intensity of disease.

Glomeruli are susceptible to a variety of inflammatory, metabolic, hemodynamic, toxic, and infectious insults. Most glomerular disease is triggered by immune attack, metabolic stress, or mechanical stress. Diverse insults can induce similar clinicopathological presentations, suggesting marked overlap among dowmstream molecular and cellular responses.

The consequences of injury at different sites within tne glomerulus can predicted from the physiological functions of the cells within the local milieu. The major sequelae of injury to the endothelium and subendothelial aspect of the GBM are

1. Recruitment of leukocytes leading to inflammatory glomerulonephritis, or
2. Perturbed hemostasis leading to thrombotic microangiopathy.

It is usual for one of these parameters to dominate; however, hybrid lesions may occur. Internal vasoconstriction and mesangial cell contraction can complicate each phenotype and thereby contribute to renal failure; Injury localized to mesangial area

typically presents as asymptomatic abnormalities of urinary sediment and mild renal insufficiency. Proteinuria dominates the clinical presentation of injury to the subepithelial aspect of GBM and visceral epithelial cells. As with mesangial injury, GFR is often mildly compranised in this setting unless there is concomitant tubulointerstitial injury. The classic pathological manifestation of parietal cell injury is crescent formation, which typically presents with acute or subacute renal failure. Crescents can be the dominant morphologic presentation of glomerular disease or complicate proliferative or membranous lesions.

IgA nephropathy

IgA nephropathy (Berger's disease, IgAN) is an immunopathologic entity characterized by deposition of granular immunoglobulin A, and frequently C3 in the glomerular mesangium.

Nephritis is present in 80% of patients and manifests as a nephritic urine sediment and moderate proteinuria. Macroscopic hematuria and nephritic – range proteinuria are uncommon.

Light – microscpic appearance can vary from mild mesangial proliferation and expression of diffuse proliferation with glomerular crescents.

The sine qua non for diagnosis is the presence of mesangial IgA deposition on immunofluorescence microscopy. IgA and C3 are also detected. Electron microscopy reveals mesangial immune deposits. Immune complexes may also be present in the peripheral glomerular capillary wall and paramesangial areas.

Minimal change disease

MCD accounts for 80% of nephritic syndrome in children younger than 16 years than 20% in adults .The peak incidence is between 6 and 8 years. Patients typically present with nephritic syndrome and benign urinary sediment. Microscopic hematuria is present in 20 to 30 %. Hypertension and renal failure are very rare.

MCD (also called nil disease, lipid nephrosis, or foot process disease) is so named because glomerular size and architecture are normal by light microscopy. Immunofluorescence studies are typically negative for immunoglobulin and C3. The findings of mesangial hypercellularity and sparse deposits of C3 and IgM portend a worse prognosis. Electronmicroscopy reveals characteristic diffuse effacement of the foot processes of visceral epithelial cells.

The etiology of MCD is uncommon, and the vast majority of cases are idiopathic. MCD occasionally develops after upper respiratory tract infection, immunizations, and atopic attacks.

In children, the urine contains albumin principally and minimal amounts of higher molecular weight proteins such as alpha 2 macroglobulin and IgG. This selective proteinuria in conjugation with foot process effacement suggests injury to podocytes loss of fixed negative charge in the glomerular filtration barrier for protein. Proteinuria is typically nonselective in adults, suggesting more extensive perturbation of membrane permeability.

Focal Segmental Glomerular Disease

The pathagnomonic morphologic lesion in FSGS is sclerosis with hyalinosis involving portions (segmental) of fewer than 50% (focal) of glomeruli on tissue section. The incidence of idiopathic (primary) FSGS had increased over the past two decades so that it now accounts for about one – third of cases of nephritic syndrome. Secondary FSGS can complicate a number of systemic diseases and sustained glomerular capillary hypertension following nephron loss from any cause.

Idiopathic FSGS typically presents as nephritic syndrome (66%) or subnephrotic proteinuria (33%) in association with hypertension, mild renal insufficiency, and abnormal urine sediment that contains red blood cells and leukocytes. Proteinuria is non selective in most cases.

Light microscopy of renal biopsy tissue reveals FSGS with entrapment of amorphous hyaline material, a process that shows a predilection for juxtamedullary glomeruli. Electron microscopy reveals evidence of damage to visceral epithelial cells.

The etiology of primary FSGS is unclear, but appears to be, at least in part, immunological.There is evidence that a circulating non immunoglobulin permeability factor, possibly a lymphokine, triggers FSGS in at least a subgroup of patients. Plasmapharesis has been employed with variable success to control the

nephrotic syndrome in this group. Secondary FSGS is a potential long-term consequence of nephron loss from any cause.

Membranous Nephropathy

This morphologic lesion is a leading cause of idiopathic nephritic syndrome in adults (30-40%) and a rare cause in children (<5%). It has a peak incidence between the ages of 30 to 50 years and a male – female ratio of 2:1. Membranous nephropathy derives its name from the characteristic light microscopic appearance on renal biopsy, namely diffuse thickening of the GBM, which is more apparent upon staining with periodic acid-schiff (PAS). Most patients (>80%) present with nephritic syndrome, proteinuria usually being nonselective. Microscopic hematuria is present in up to 50% of cases. Hypertension is documented in only 10 to 30% of patients at the outset but is common later in patients with progressive renal failure. Serologic test such as anti nuclear antibody, ANCA, anti-GBM antibody,cryoglobulin titers and complement levels are normal in the idiopathic form

Light microscopy of the renal biopsy section reveals diffuse thickening of the GBM without evidence of inflammation or cellular proliferation. Immunofluorescence reveals granular deposition of IgG, C3 and the terminal components of compliment along the glomerular capillary wall.

The pathogenesis of idiopathic human membranous glomerulopathy is incompletely understood .The presence of electron – dense immune deposits that contain IgG and C3 suggest an immune process. About one third of adult membranous nephropathy occurs in association with systemic diseases such as SLE, infections such as hepatitis B, malignancy and drug therapy with gold and penicillamine.

Membrano Proliferative Glomerulonephritis

This morphologic entity, also known as mesangiocapillary glomerulonephritis, is characterized by thickening of the GBM and proliferation changes on light microscopy. Two major types are identified; both are characterized by a diffuse increase in mesangial cellularity and matrix, and by thickening and reduplication of the GBM such that the lobular pattern of the glomerular tuft is exaggerated. The hallmark of type I MPGN is the presence of subendothelial and mesangial deposits on electron microscopy that contain C3 and IgG or IgM; rarely IgA deposits are demonstrated by immunofluorescence microscopy.The hallmark of type II MPGN (dense deposit disease) is the presence of electron-dense deposits within the GBM and other renal basement membranes that stain for C3, but little or no immunoglobulin.

Most patients with type I MPGN present with heavy proteinuria or nephritic syndrome, active urinary sediment, and normal or mildly impaired

GFR.C3 levels are usually depressed and C1q and C4 levels are borderline or low. Type MPGN is an immune-complex glomerulonephritis and can be associated with a variety of chronic infections, systemic immune complex disease and malignancies

Type II MPGN can also present with proteinuria and nephritic syndrome; however, some patients present with nephritic syndrome, RPGN or recurrent macroscopic hematuria. Type II MPGN is an autoimmune disease in which patients have an IgG autoantibody, termed C3nephritic factor, that binds to C3 convertase, the enzyme that metabolizes C3, and renders it resistant to inactivation .Type II MPGN runs a variable course; the GFR remains stable in some patients and declines gradually to ESRD over five to ten years in others.

Mesangio Proliferative Glomerulonephritis

In 5 to 10% of patients with idiopathic nephrotic syndrome, renal biopsy reveals a diffuse increase in glomerular cellularity, predominantly due to proliferation of mesangial and endothelial cells, and infiltration by monocytes. Findings on immunofluorescence microscopy vary and include deposits of IgA, IgG, IgM, and /or complement, or absence of immune reactants. It is likely that this morphologic entity is, in fact, a heterogeneous group of diseases that includes atypical forms of MCD and FSGS and milder or resolving forms of the immune-complex and pauci-immune glomerulopathies described above under nephritic

syndrome and RPGN. In keeping with the heterogeneity of this diagnosis, the prognosis is variable. In general, persistent nephritic –range proteinuria signals a poor prognosis, with many patients progressing to ESRD over 10 to 20 years despite immunosuppressive therapy.

Renal Amyloidosis

Amyloidosis is classified according to the major component of its fibrils. There is substantial overlap in renal clinicopathologic presentations of amyloid (AL) and amyloid A (AA). Glomeruli are involved in 75 to 90% of patients usually in association with involvement of other organs. The clinical correlate of glomerular amyloid deposition is nephrotic-range proteinuria. In addition, >50% of patients have impaired glomerular filtration at diagnosis. Hypertension is present in about 20 to 25%. Renal size is usually normal or slightly enlarged. A minority of patients present with renal failure due to amyloid deposition in renal vasculature with fanconi's syndrome, nephrogenic diabetes insipidus, or renal tubular acidosis due to involvement of the tubulo interstitium. Rectal biopsy and abdominal fat pad biopsy reveal amyloid deposits in about 70% of patients and may obviate the need for renal biopsy.

Renal biopsy gives a very high yield if there is clinical evidence of renal involvement. The earliest pathologic changes are mesangial expansion by amorphous hyaline material and thickening of the GBM. Further amyloid

deposition results in the development of large nodular eosinophilic masses. When stained with congo red, theses deposits show apple-green birefringence under polarized light. Electron microscopy the characteristic non-branching extra cellular amyloid fibrils of 7.5 to 10 nm in diameter. Tubulointerstitial and vascular deposits of amyloid are also seen and may occasionally be more predominant than glomerular deposits.

Lupus Nephritis

Nephritis is usually the most serious manifestation of SLE. Since nephritis is asymptomatic in most lupus patients, urinalysis should be ordered in any person suspected of having SLE. The classification of lupus nephritis is primarily histologic. Renal biopsy is useful in planning current near future therapies. Patients with dangerous proliferative forms of glomerular damage usually have microscopic hematuria and proteinuria (>500 mg per 24 h); approximately one-half develop nephrotic syndrome, and must develop ESRD within 2 years of diagnosis. Therefore, aggressive immunosuppression is indicated (usually systemic immunosuppression plus a cytotoxic drug), unless damage is irreversible. Usually 20% of the patients with DPGN die or develop ESRD within 10 years of diagnosis. Such individuals require aggressive control of SLE and of complications of renal disease and therapy. A small proportion of SLE patients

with proteinuria (usually nephrotic) have membranous glomerular changes without proliferation on renal biopsy. There out come is better than for those with DPGN, but proteinuria is less likely to improve. Lupus nephritis tends to be an ongoing disease, with flares requiring re-treatment over many years. For most people with lupus nephritis, accelerated atherosclerosis becomes important after several years of disease; attention must be given to control of blood pressure, hyperlipidemia, and hyperglycemia.

In renal biopsies, the pattern of injury is important in diagnosis and in selecting the best therapy. The World Health Organisation (WHO) has classified lupus nephritis as

Grade I – no histological changes

Grade II – proliferative changes confined to the mesangium

Grade III – proliferative changes in tufts of 10 to 50 % of glomeruli; higher proportions of glomeruli affected suggest worse prognosis

Grade IV – diffuse proliferative glomerulonephritis (DPGN) affecting >50% of glomeruli.

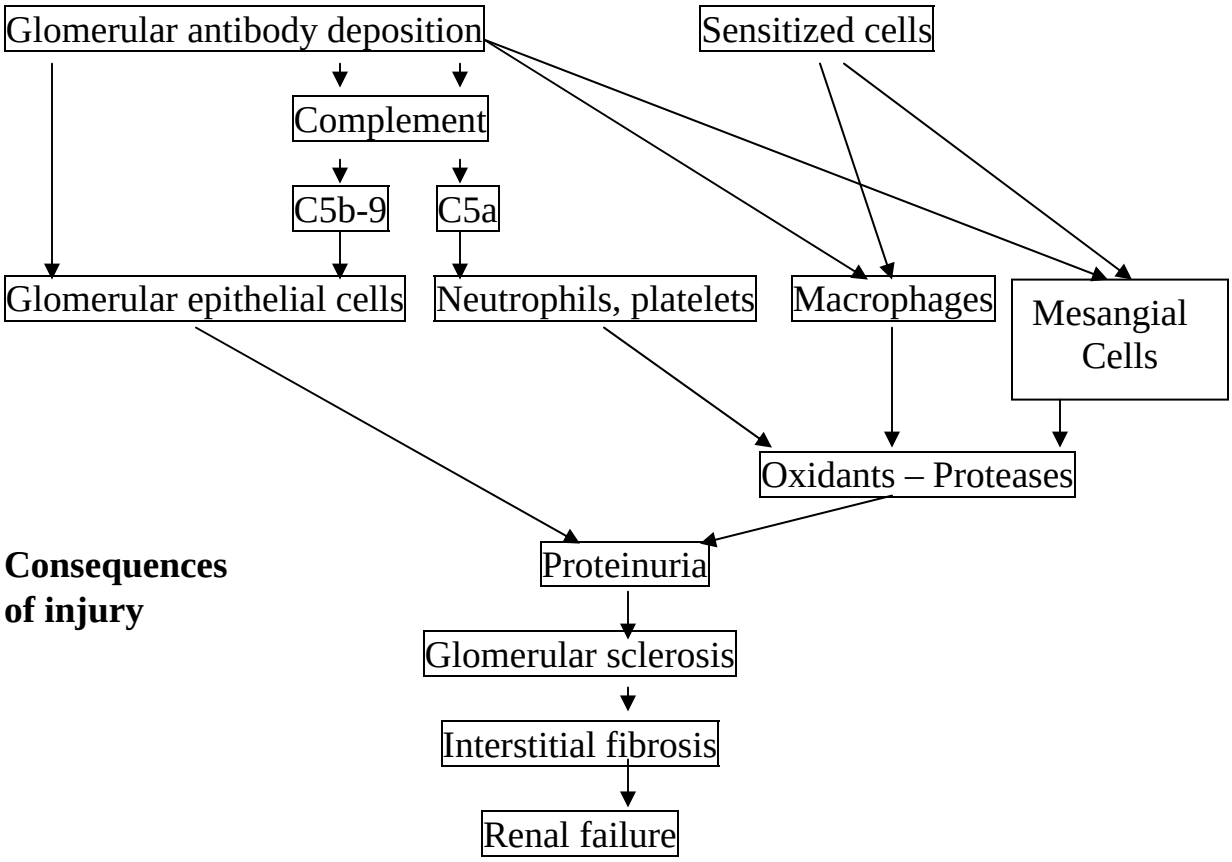
Grade V - predominantly membranous changes with various degrees of proliferation

Grade VI – end stage, scarred glomeruli.

In addition, pathologist report the extend of inflammatory (potentially reversible) and chronic (irreversible scarring in glomeruli, renal tubules, and blood vessels) changes.

In general, treatment for lupus nephritis is not recommended in patients with class I or II disease or with extensive irreversible changes. In contrast, aggressive immunosuppression is recommended for patients with class III, IV or V inflammatory proliferative lesions because the majority of those individuals, if untreated, develop end-stage renal disease (ESRD) within 2 years.

Mechanisms of glomerular injury



In the normal person, urinary protein excretion is less than 150 mg per day (with most subjects being under 100 mg per day) and consists mostly of filtered plasma proteins (60%) and tubular Tamm-Horsfall proteins (40%). The main plasma protein in the urine is albumin, constituting about 20% of the total normal daily

protein excretion. In normal subjects the daily amount of albumin is less than 20 mg (15µg/min).

Proteinuria usually reflects an increase in glomerular permeability for normally non-filtered plasma macromolecules such as albumin. A 24-hour urine collection containing more than 150 mg of protein is abnormal. Significant proteinuria is suspected when a dipstick test of the urine is persistently positive for protein. In such a situation the daily protein excretion will usually exceed 300-500 mg per day. Since the dipstick method can detect urine protein concentration of as little as 30 mg/dL, a very concentrated urine specimen might test positive for protein even though the quantitative amount of proteinuria is less than 150 mg/day.

The dipstick method for detection of proteinuria relies on a color change of the indicator dye—a reaction primarily dependent on the amount of albumin in the urine. The sulfosalicylic acid (SSA) method of protein detection may be useful in certain situations, but is generally not readily available in most clinical situations. The SSA method detects proteinuria by acid precipitation and detects any type of proteins in the urine

Four important qualifications to the foregoing definitions are needed: 1.) Both dipstick and SSA will record false positive results for protein in the presence of

radiocontrast agents.2.) Since the dipstick method is most sensitive for detection of albumin, disease states in which the proteinuria is mainly composed of heavier plasma proteins such as immunoglobulins or their light-chains (multiple myeloma or plasma cell dyscrasias) may be associated with a negative or weakly positive dipstick reaction for protein. The SSA method will give a strongly positive reaction and subsequent analysis of the urine for immunoglobulins or light-chains will verify that the proteinuria is due to proteins other than albumin. 3.) Dipstick and SSA will both detect urinary lysozymes that are increased in production and excretion in some patients with acute monocytic leukemia. 4.) In early diabetic nephropathy, persistent excretion of increased amounts of albumin in the range of 30-300 mg daily is called microalbuminuria. Standard dipstick methodology might fail to detect such small but clinically important amounts of albuminuria. Hence using a specific assay for urinary albumin is the more sensitive and recommended technique.

Microalbuminuria is defined by the presence of >30 and <300 mg of albuminuria daily. Since 24-hour urine collections are difficult to obtain, the measurement of the albumin to creatinine concentration in an untimed random urine specimen is used. The albumin to creatinine concentration of >30 mg per gram of creatinine correlates very well with a 24-hour urine albumin measurement. Its detection in Type I diabetes mellitus is the earliest clinical evidence of diabetic nephropathy. Transient increases in urinary albumin excretion may be seen in short-term

hyperglycemia, exercise, urinary tract infections, marked hypertension, heart failure, and acute febrile illnesses. There is also diurnal variation in urinary albumin excretion. Confirmation of microalbuminuria requires verification on 2 or 3 collections over 3 to 6 months. Transient and clinically insignificant dipstick proteinuria might occur in a variety of clinical states including febrile illnesses, following vigorous exercise, in congestive heart failure, and exposure to cold. Transient proteinuria might be seen in 4% of men and 7% of women on a single examination. In screening studies of asymptomatic healthy individuals, dipstick proteinuria on 2 consecutive occasions has been reported in from 0.5-5%. In the same studies, the positive predictive value of proteinuria identifying serious urogenital disease was 0-11%. Orthostatic proteinuria occurs primarily in older adolescents and is characterized by increased protein excretion in the upright position and normal protein excretion in the supine position (ie, as found in a urine sample taken early in the morning after the subject has been lying down). Orthostatic proteinuria is a benign condition. Persistent proteinuria, especially in clinical illnesses, or when accompanied by other urinary abnormalities, such as hematuria, proteinuria, or bacteruria, deserves further investigation. In one study of adults, IgA nephritis, membranous nephropathy, and focal and segmental glomerulosclerosis were the most common histologic diagnoses in patients with asymptomatic proteinuria and/or hematuria.

There are 3 basic types of proteinuria—glomerular, tubular, and overflow. The glomerular filtration barrier is composed of the endothelial cell, the basement membrane, and the epithelial cell foot processes. Proteinuria occurring in glomerular disease is due to increased filtration of albumin and other macromolecules across the glomerular basement membrane. This occurs because of an alteration in both the charge selectivity and size selectivity of the glomerular barrier.

Glomerular proteinuria

Normally the basement membrane and endothelial cells possess a negative charge. Plasma albumin, which also possesses a negative charge, is repelled by the normal negative charge on the basement membrane and the intact endothelial cells. Circulating IgG has a neutral or positive charge and is not restricted by a negative charge on the basement membrane. Rather, immunoglobulins are restricted by the size selective barrier of the membrane and the epithelial slit diaphragm located across the spaces between the epithelial foot processes

In glomerular disease, the injury to the glomerular basement membrane causes proteinuria due to a loss in negative charge as well as from an increase in the number of larger non-selective pores. Glomerular diseases are also accompanied by disruption and loss of the epithelial foot process covering of the basement membrane. It appears that the increased protein leakage occurs especially at the sites of this epithelial alteration.

Tubularproteinueia

Low molecular weight molecules such as B2 microglobulin, amino acids, and immunoglobulin light chains have a molecular weight of about 25000 (albumin is 69000). These smaller proteins are easily filtered across the basement membrane and then completely reabsorbed by the proximal tubular cells. A variety of diseases that produce tubular and interstitial injury impair the tubular reabsorption of these molecules. Some glomerular diseases are also accompanied by tubular injury and tubular proteinuria. Standard dipstick methods will not detect these proteins. Specific urinary measurements of B2 microglobulin are quite sensitive for any tubular injury, but they are not specific for any disease.

Overflowproteinuria

Increased excretion of low molecular weight proteins might be seen in states where there is significant increased production of these proteins, as in multiple myeloma. The proteinuria results from the fact that the amount of these proteins filtered exceeds the reabsorptive capacity of the proximal tubule.

Most patients with proteinuria have no signs or symptoms from the proteinuria. In states of heavy (nephrotic range) proteinuria exceeding 3 g daily, the patient might report foamy urine and might demonstrate edema. The foamy urine is due to increased lipid in the urine, which alters the surface tension of the urine. Lipiduria is caused by the filtration of lipoproteins across the damaged glomerular barrier. On urine microscopy lipiduria might appear as free fat, or as fat droplets in tubular

cells or casts where they are referred to as oval fat bodies or fatty casts respectively. Edema, which frequently accompanies nephrotic range proteinuria, is caused by reduction of plasma oncotic pressure due to reduced plasma albumin. Hypoalbuminemia is the result of increased glomerular losses and defective synthesis of albumin. At times the hypoalbuminemia and loss in plasma oncotic pressure produce true intravascular volume depletion resulting in hypotension and pre-renal acute renal failure. The loss of albumin stimulates the liver synthetic activity, which also contributes to increased lipoprotein production and hyperlipidemia.

In addition, a careful microscopic exam of recently voided centrifuged urine should be performed. The presence of bacteria and leukocytes will suggest urinary infection. The presence of leukocytes and leukocyte casts in the absence of bacteruria suggests interstitial nephritis. The presence of dysmorphic erythrocytes and erythrocyte casts suggests a nephritic syndrome such as an acute or subacute glomerulonephritis, IgA nephropathy, or membranoproliferative glomerulonephritis. The presence of fatty casts or oval fat bodies suggests that the patient has a glomerular disease associated with nephrotic syndrome or nephrotic range proteinuria. In patients with a benign urine sediment, acute renal failure and slightly positive dipstick for proteinuria, one should perform a sulfosalicylic acid (SSA) test of the urine. A strongly positive SSA and weakly positive dipstick proteinuria should lead one to suspect light chains in the urine and a diagnosis of

some type of plasma cell dyscrasia. Quantification of protein excretion may be performed on a complete 24-hour urine specimen or by determining the protein:creatinine ratio in a random urine specimen. The protein:creatinine ratio has been validated and shown to compare favorably with complete 24-hour collection. Thus, a protein:creatinine ratio of less than 1 and greater than 3 is consistent with 24-hour protein excretions of less than one gram and greater than 3.5 g respectively. This method is also easier and likely to be more accurate since it does not require a 24-hour collection of urine.

Quantification of protein excretion allows one to begin to differentiate among the various renal disorders causing proteinuria. Diseases may be grouped into those with less than 1-2 grams/day, and greater than 3.5 grams daily. Patients with less than 1-2 grams of proteinuria usually have tubulointerstitial disease, nephrosclerosis, polycystic kidney disease, orthostatic proteinuria, or benign glomerular disease such as IgA nephritis. Proteinuria of greater than 3.5 g per day is due to glomerular diseases.

RENAL BIOPSY

While the procurement of kidney tissue allows a detailed morphological analysis, this is not always indicative of the patient's diagnosis. This holds true particularly in patients in whom the renal manifestations are secondary to systemic disease. The prime aims of performing a renal biopsy are

1. A histological determination of the type and severity of renal disease
2. Deciding whether specific treatment (steroids, immunosuppression, plasmapheresis), could be beneficial, and
3. Estimating prognosis and potential for reversibility of the lesion

HISTORICAL PERSPECTIVE:

In 1923 Gywn first performed an open renal renal biopsy in patients with nephrotic syndrome. The first percutaneous renal biopsy is attributed to Ball, used an aspiration device to help diagnose a hypernephroma.

PERCUTANEOUS BIOPSY PROCEDURE:

Biopsies are usually done in the morning after an over night fast. An intravenous access is placed. The patient lies on his abdomen on a firm table. With the patient in the prone position the operator working from the right hand side of the bed, the left kidney is nearest to the operator.

Following localization with ultrasonogram, the skin site is marked and the surrounding area surgically cleansed, draped and infiltrated with a local anesthetic. After anesthetizing the deeper tissues, the biopsy needle is inserted through a

small skin incision. The movements of the needle in relation to the patients breathing serve to guide the tip in relation to the kidney. On respiration when, the kidney moves independently under the tip of the needle, the needle is superficial to renal parenchyma. On penetrating the capsule, the needle will swing with a pendular motion, towards the head on inspiration and returning caudally on expiration. The needle may be seen to jerk free of the capsule, again assuming a motion independent of respiration. At this point the needle should be inserted downwards a few millimeters entering the renal cortex. Once inside the cortex, the needle will be fixed within the kidney and again on respiration will move in unison with it. On the cessation of breathing, the needle will also be seen to pulsate in accordance with the periodic flow of blood to the kidneys.

Once the needle is properly positioned in the renal cortex with the patient holding his breath, the specimen is cut and the needle withdrawn out. The inspection of the tissue pieces with a dissecting microscope for glomerular tufts ensues the adequacy of the biopsy specimen. The presence of at least six glomeruli for light microscopy is considered as a successful biopsy.

COMPLICATIONS:

The most common complication encountered is hematuria. Microscopic hematuria occurs in virtually all patients, whereas gross hematuria is seen in 3-16% of biopsies. Perinephric hematomas are a common occurrence. Clinically significant hematomas are encountered in 0.2 to 2% of biopsies, manifesting as a flank mass and changes in vital signs in association with a fall in hematocrit. Other

complications include: ileus, laceration of the liver, intestine, gall bladder, subcostal and visceral arteries, Pneumothorax; and puncture of the renal pelvis resulting in urinoma. Infections are uncommon except in case of pyelonephritis.

CONTRAINDICATIONS:

1. Solitary kidney / a sole functioning kidney.
2. Uncontrolled hypertension.
3. Hemorrhagic diathesis.
4. Isolated large renal cyst.
5. Uncooperative patient.

MATERIALS AND METHODOLOGY

MATERIALS AND METHODOLOGY

This is a prospective study of fifty consecutive patients attending the nephrology clinic in The Stanley medical college hospital, Chennai in whom ultrasonogram guided renal biopsy were done in patients with the following criteria

INCLUSION CRITERIA:

1. Patients aged more than twelve years
2. Proteinuria more than 1 gms / 24 hours

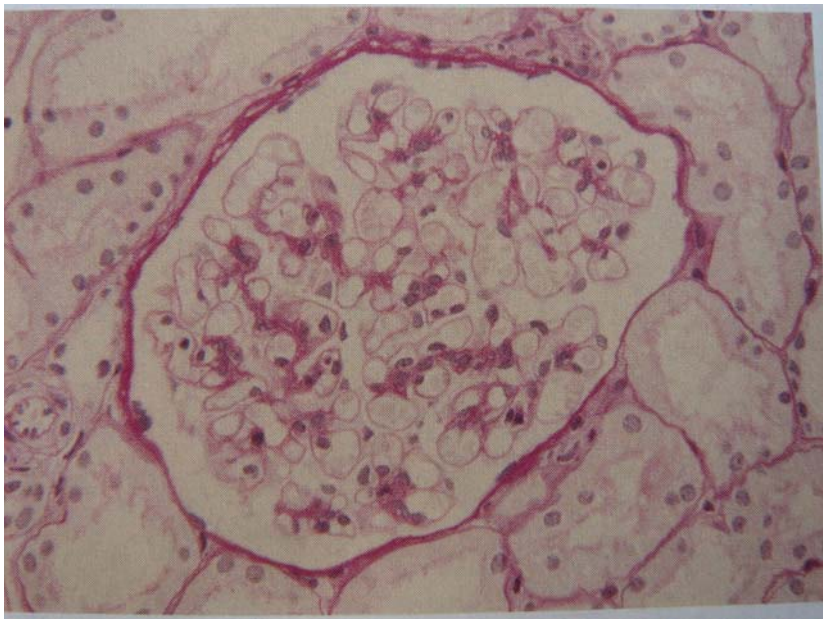
EXCLUSION CRITERIA:

1. All known cases of diabetes mellitus
2. All known cases of systemic hypertension
3. All known cases with contracted kidneys proved by ultrasonogram
4. Post transplant patients
5. Cases of nephrotic syndrome responding to immunosuppressive therapy

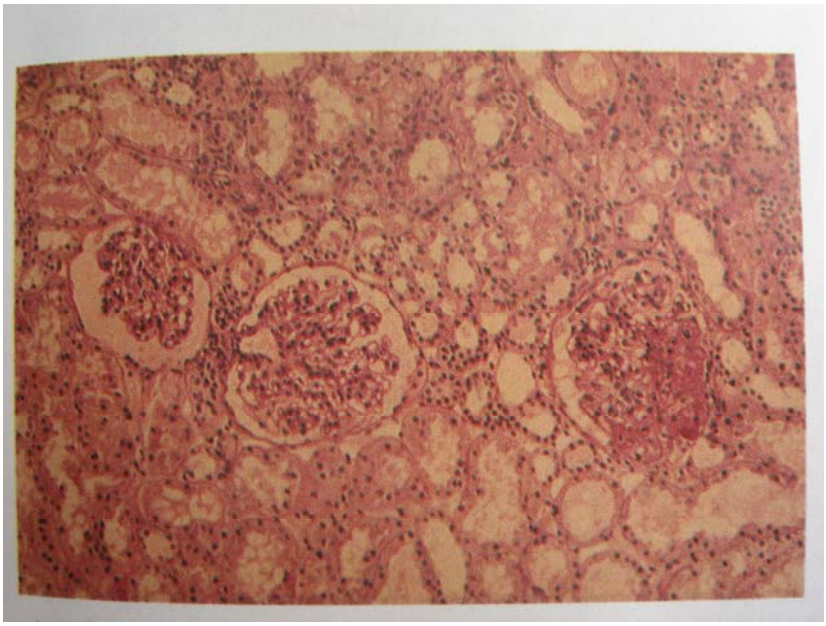
Renal biopsy helps in establishing accurate diagnosis, identifying any reversible pathology, helps in devising appropriate management plan for the patients and is very useful in understanding the histological nature of the disease The incidence of glomerular disease had increased dramatically in last decade and

the pattern of glomerular pathology is changing dynamically with time .The type of glomerular disease varies in different geographies and among different age group of patients The amount of proteinuria, renal insufficiency, hypertension, and microscopic hematuria differ in different histopathological types and in different age groups .Hence all these parameters and the histopathological type of glomerular disease have prognostic implications in patients with glomerular disease Renal biopsy was performed in patients with glomerular disease (proteinuria <1 gms/24 hrs) attending the nephrology clinic from august 2004 to august 2006 All the biopsy specimen were evaluated by light microscopy and immunofluorescent staining. Laboratory investigations including twenty four hour urinary proteinuria, serum creatinine, hemoglobin, total leukocyte count, differential count, erythrocyte sedimentation rate, serum proteins, serum calcium, serum cholesterol, ultrasonogram of kidneys were done along with the histopathological analysis of kidney biopsy

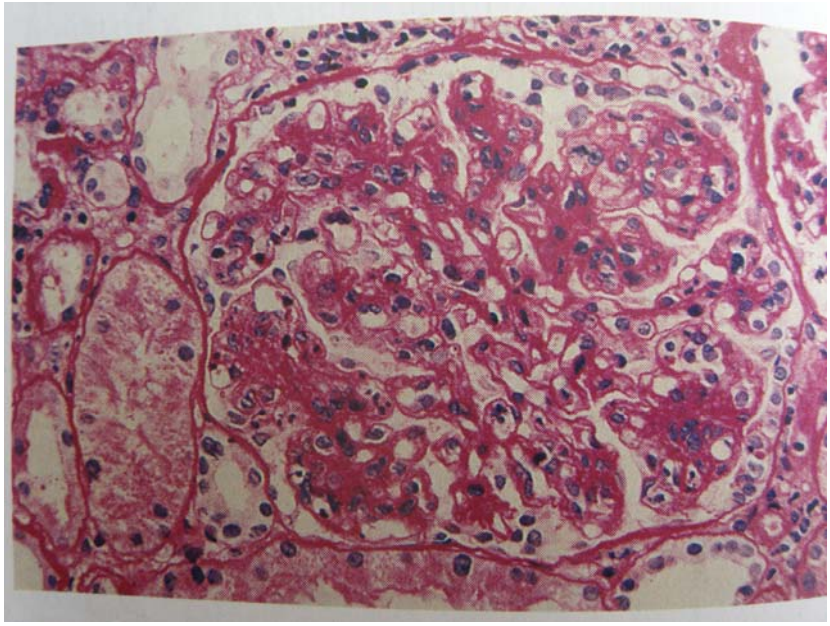
MINIMAL CHANGE DISEASE - PAS – STAINING



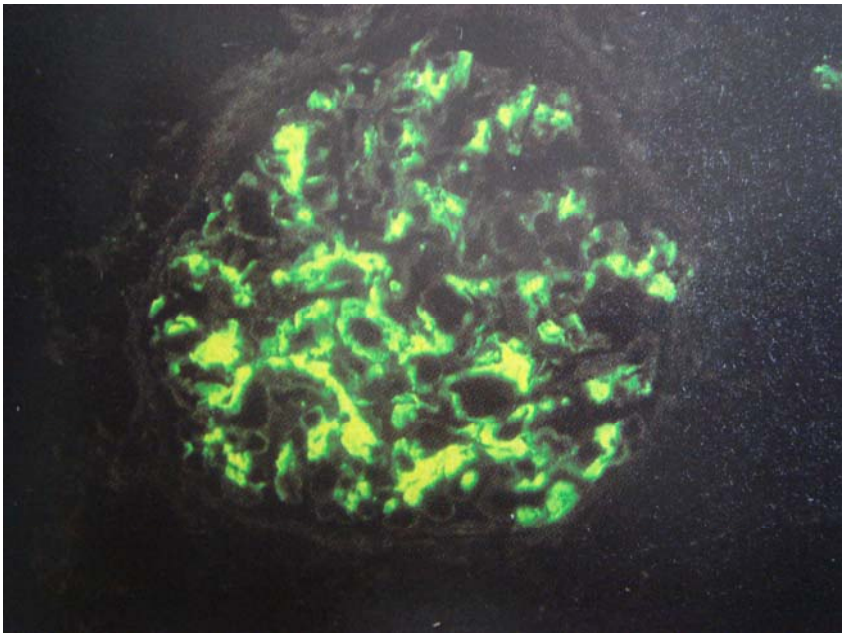
FSGS – PAS STAIN - LOW POWER VIEW



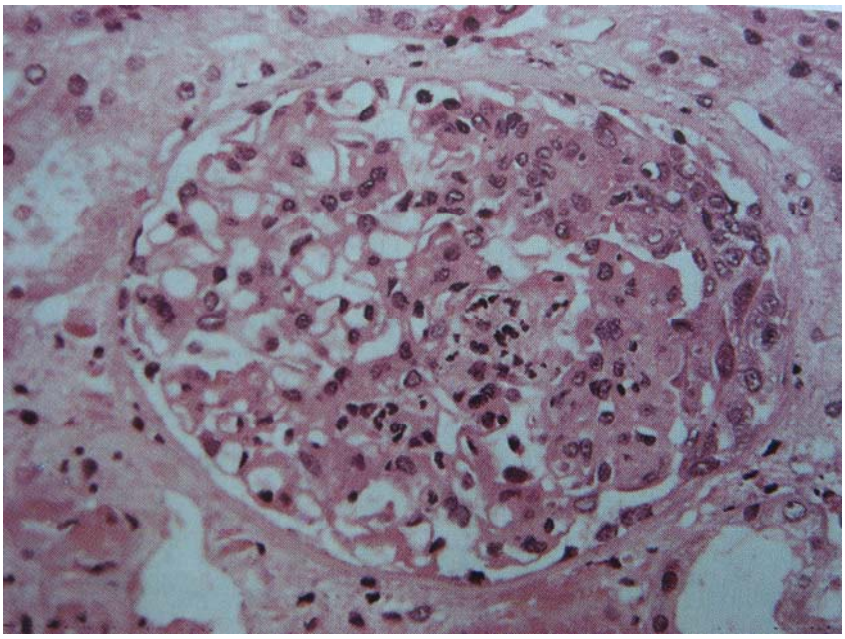
FSGS – PAS STAINING - HIGHPOWER VIEW



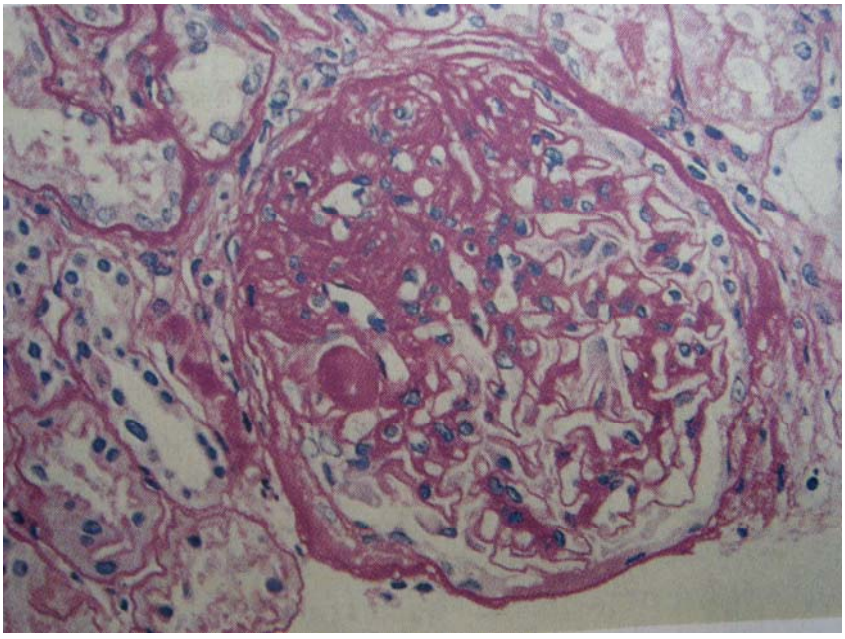
IgA NEPHROPATHY – IMMUNOFLUORESCENCE STAINING



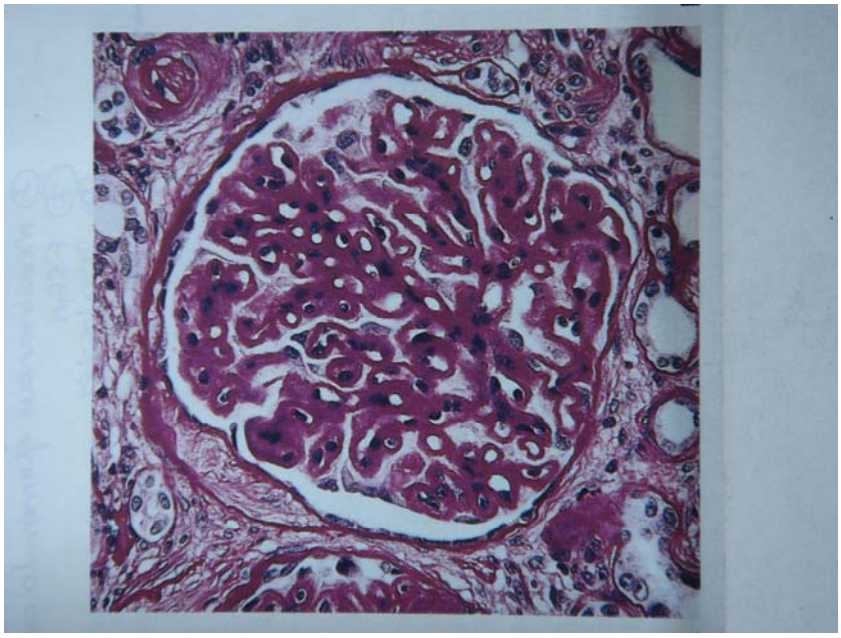
FOCAL GLOMERULONEPHRITIS IN LUPUS ERTHEMATOSUS



FSGS – HIGH POWER VIEW SHOWING HYALINE MASS AND LIPID SCLEROTIC AREA



MEMBRANOUS GLOMERULONEPHRITIS PAS STAINING



RESULTS AND ANALYSIS

AGE SEX DISTRIBUTION

Age group (in years)	Male (%)	Female (%)	Total (%)
12- 20	**7 (14%)**	**7 (14%)**	**14 (28%)**
21- 40	**10 (20%)**	**20 (40%)**	**30 (60%)**
41-60	**1 (2%)**	**5 (10%)**	**6 (12%)**
Total	**18 (36%)**	**32 (64%)**	**50 (100%)**

The age sex distribution shows that

The mean age of all the patients – 27.5 years

Patients in age group 12- 20 – 18;

Males – 7, Females – 7. Mean age – 14.85 years.

Patients in age group 21 -40 years – 30;

Males -10, Females – 20.Mean age – 30.6 years

Patients in the age group 41- 60 – 6;

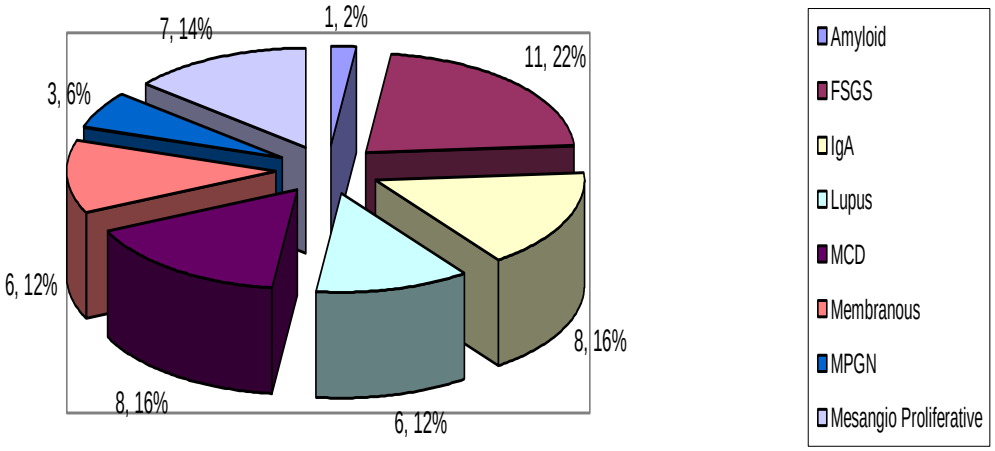
Males -1, Females – 5, Mean age – 44.5

FREQUENCEY OF THE HISTOPATHOLOGICAL TYPES

Histopathological type	Number of cases (n)	Number of cases (%)
Focalsegmental glomerulosclerosis	**11**	**22**
IgA nephropathy	**8**	**16**
Minimal change disease	**8**	**16**
Mesangioproliferative glomerulonephritis	**7**	**14**
Membranous nephropathy	**6**	**12**
Lupus nephritis	**6**	**12**
Membranoproliferative glomerulonephritis	**3**	**6**
Amyloidosis	**1**	**2**

Among the 50 patients studied Focal segmental glomerulosclerosis (FSGS) was found in 11,IgA nephropathy and minimal change disease (MCD) in 8; Mesangiproliferative glomerulonephritis in 7, Membranous nephropathy and Lupus nephritis in 6, Membranoproliferative glomerulonephritis (MPGN) in 3 and Amyloidosis in 1 patient.

Histopath Types

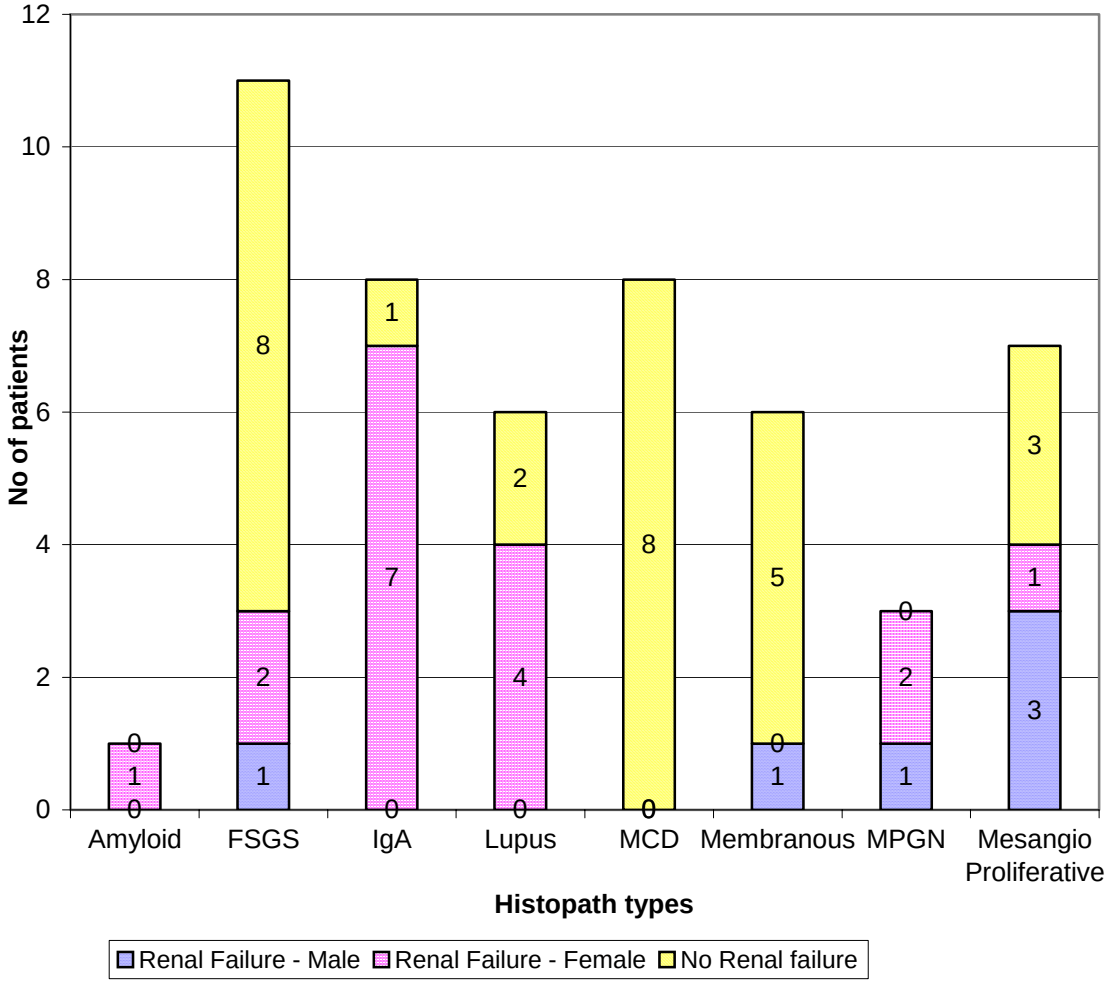


INCIDENCE OF RENAL INSUFFICIENCY

HISTOPATHOLGICAL TYPE	TOTAL PATIENTS	PATIENTSWITH RENAL INSUFFICIENCY (%)
Membranoproliferative glomerulonephritis	**3**	**3(100)**
Amyloidosis	**1**	**1(100)**
IgA nephropathy	**8**	**7(87.5)**
Lupus nephritis	**6**	**4(66.66)**
Mesangioproliferative glomerulonephritis	**7**	**4(57.14)**
Focalsegmental glomerulonephritis	**11**	**3(27.27)**
Membranous nephropathy	**6**	**1(16.66)**
Minimal change disease	**8**	**0(0)**

The incidence of renal insufficiency was common in patients with MPGN(100%) and amyloidosis (100%) , followed closely by FSGS (87.5%), lupus nephritis (66.66%), mesangioproliferative glomerulonephritis (57.14%), FSGS (27.27%) and membranous nephropathy (16.66%).

Incidence of Renal Failure



SEVERITY OF RENAL INSUFFICIENCY IN VARIOUS

HISTOPATHOLOGICAL TYPES

HISTOPATH TYPE	S.CREATININE 1.5 – 2.9 mg/dl	S.CREATININE 3- 4.9 mg/dl	S.CREATININE >5.0 mg / dl	TOTAL
AMYLOIDOSIS	0	1	O	1
FSGS	2	1	0	3
IgAN	3	3	1	7
LUPUS NEPHRITIS	4	0	0	4
MCD	0	0	0	0
MEMBRANOUS NEPHROPATHY	1	0	0	1
MPGN	0	1	2	3
MESANGIO PROLIFERATIVE	4	0	0	4
TOTAL	14	6	3	23

Of the 23 patients with renal insufficiency, number of patients with

Serum Creatinine 1.5 to 2.9 mg/dl – 14

Serum Creatinine 3.0 to 4.9 mg/dl – 6

Serum Creatinine > 5 mg/dl - 3

INCIDENCE OF HEMATURIA

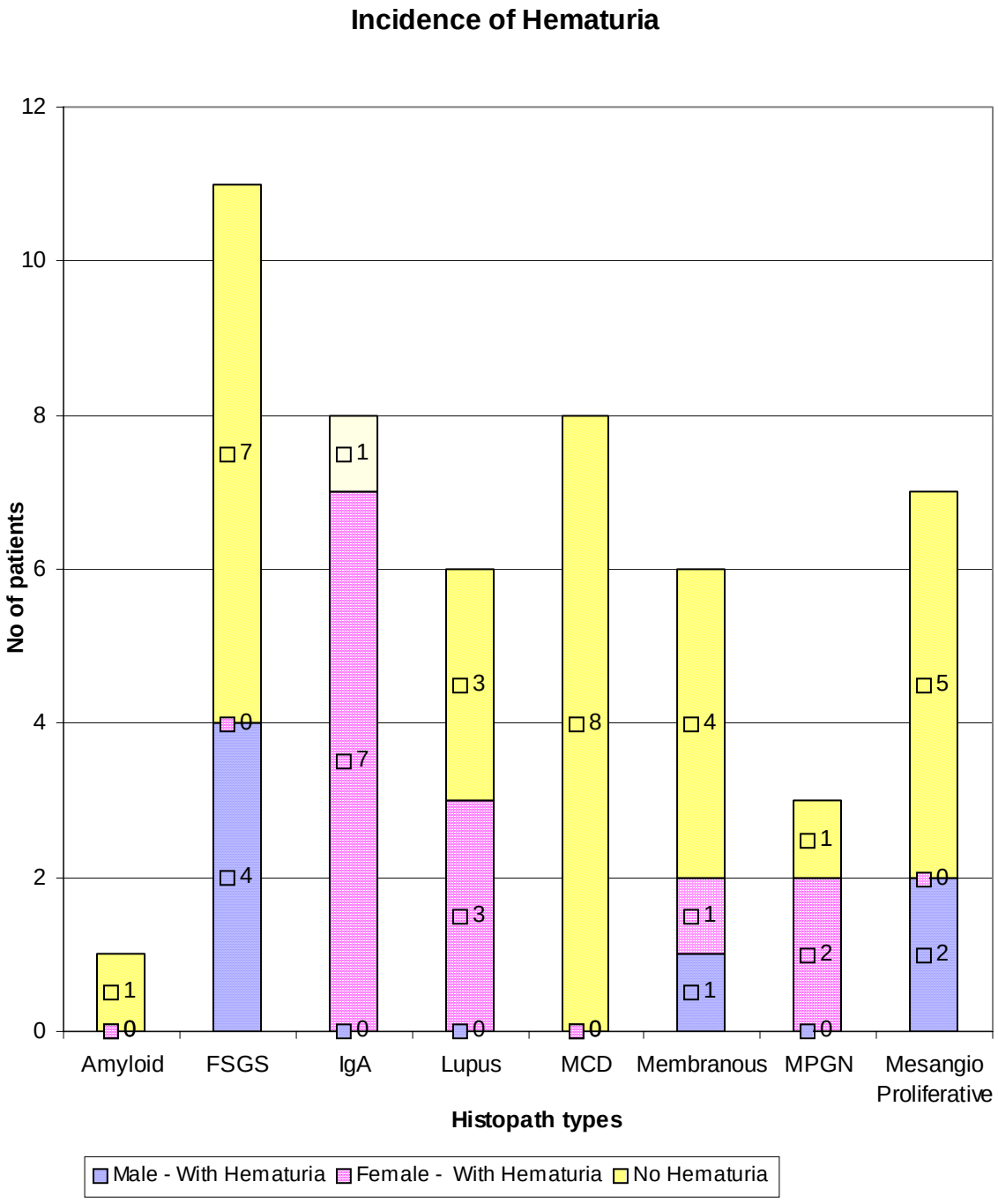
Histopathological types	Patients with microscopic hematuria (>3 RBC/HPF)		
	Males	Females	Total
IgAN	0	7	7
FSGS	4	0	4
Lupus nephritis	0	3	3
MPGN	0	2	2
Membranous GN	1	1	2
MesangioproliferativeGN	2	0	2
MCD	0	0	0
Amyloidosis	0	0	0

Total number of patients with microscopic hematuria – 20

Number of males - 7

Number of females – 13

7 patients with IgAN, 4 with FSGS, 3 with lupus nephritis, and 2 with MPGN, membranous nephropathy, mesangioproliferative glomerulonephritis each had microscopic hematuria.


Incidence of Hematuria
12
10
8
6
4
2
0
No of patients
1
0
7
4
1
7
0
3
3
0
8
0
4
1
1
1
2
0
5
0
2
Amyloid
FSGS
IgA
Lupus
MCD
Membranous
MPGN
Mesangio Proliferative
Histopath types
Male - With Hematuria
Female - With Hematuria
No Hematuria

INCIDENCE OF HYPERTENSION

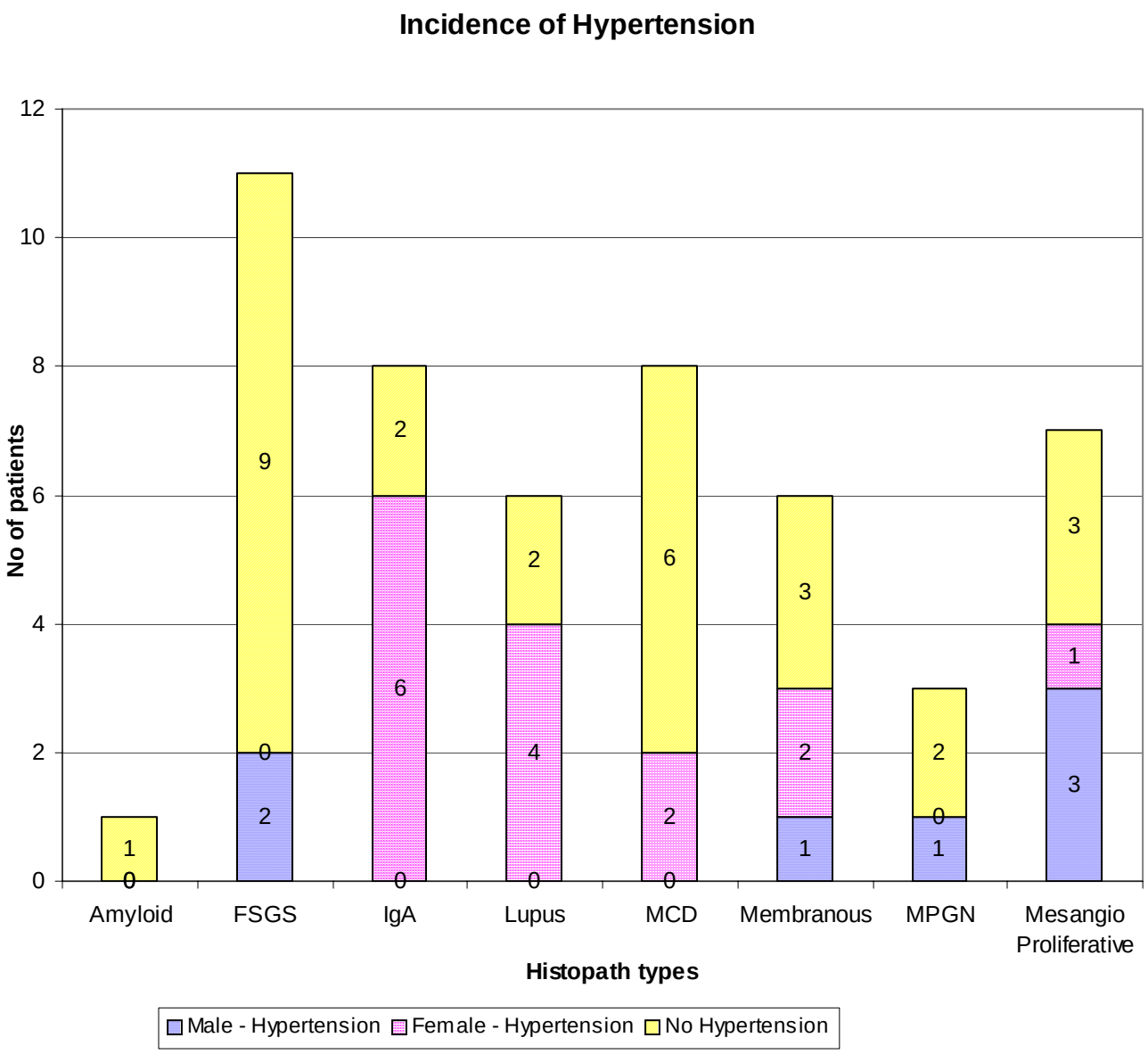
Histopath type	Patients with Hypertension (BP>140/90 mm Hg)		
	Males	Females	Total
IgAN	0	6	6
Lupus nephritis	0	4	4
MesangioproliferativeGN	3	1	4
Membranous GN	1	2	3
MCD	0	2	2
FSGS	2	0	2
MPGN	1	0	1
Amyloidosis	0	0	0

22 patients of the total 50 patients studied had Hypertension

Males with Hypertension – 7

Females with Hypertension – 15

6 patients with IgAN, 4 with Lupus nephritis and mesangioproliferative glomerulonephritis each; 3 with membranous nephropathy, 2 with MCD and FSGS each; and 1 with MPGN had Hypertension.


Incidence of Hypertension
No of patients
12
10
8
6
4
2
0
1
0
2
0
9
0
6
2
0
4
2
0
2
6
0
2
3
1
2
1
0
2
1
3
1
3
Amyloid
FSGS
IgA
Lupus
MCD
Membranous
MPGN
Mesangio Proliferative
Histopath types
Male - Hypertension
Female - Hypertension
No Hypertension

AGE WISE DISTRIBUTION OF THE STAGES OF HYPERTENSION

AGE GROUP In years	**STAGE 1** BP (140-159/90-99) mmHg	**STAGE 2** BP (>160/100) mmHg	**TOTAL**
12 – 20	**1**	**1**	**2**
21-40	**11**	**6**	**17**
40-60	**1**	**2**	**3**
TOTAL	**13**	**9**	**22**

Hypertension is more common in the age group of 21 to 40 years – 11 patients

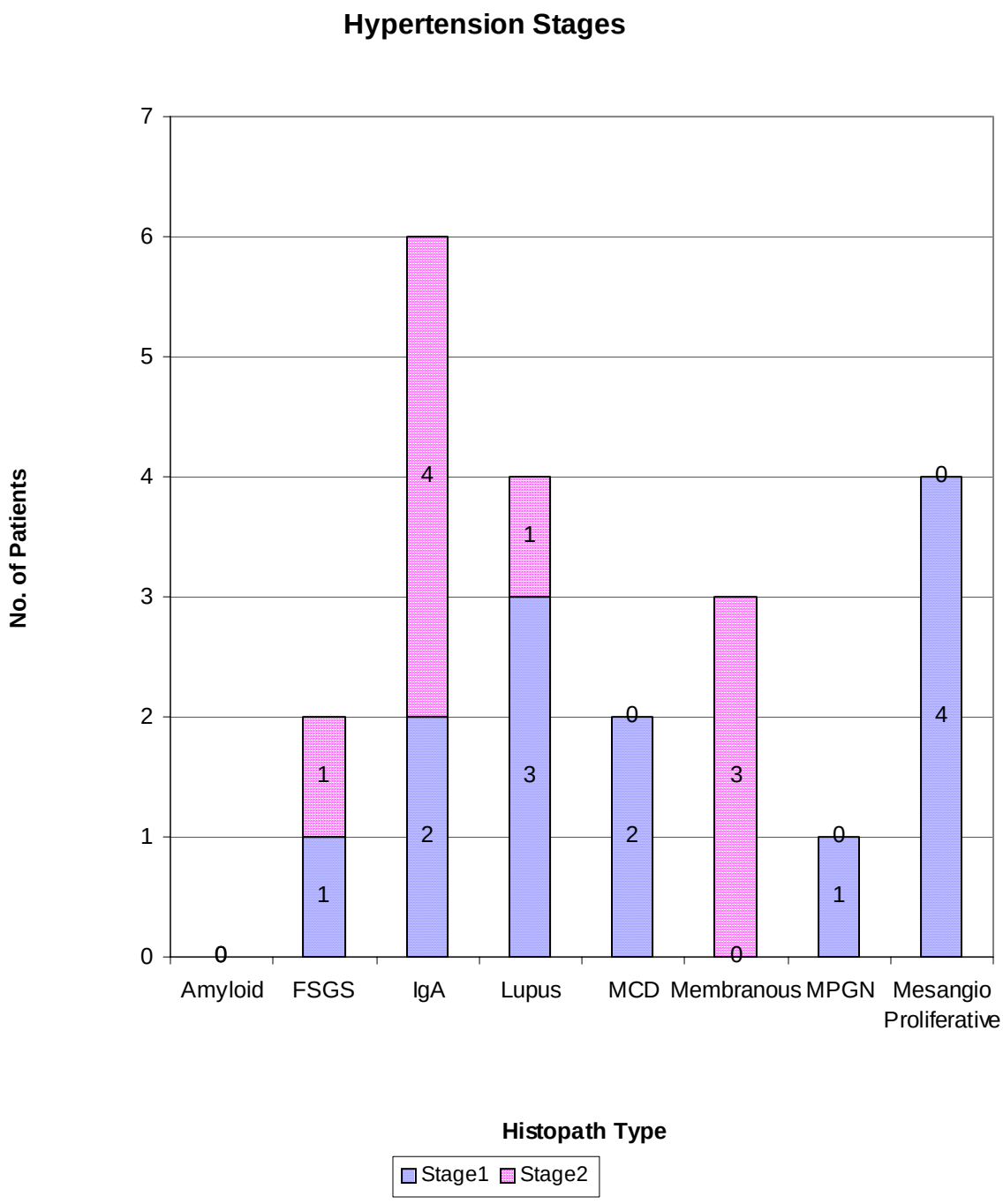
STAGE I HYPERTENSION – BP 140-159/90-99 mmHg

STAGE II HYPERTENSION – BP >160/100 mmHg

Of the 22 patients with hypertension, number of patients with

Stage I hypertension – 13(59.09%)

Stage II hypertension – 9(40.09%)


Hypertension Stages
No. of Patients
7
6
5
4
3
2
1
0
0
1
1
2
4
3
1
2
0
3
0
0
1
0
4
0
Amyloid
FSGS
IgA
Lupus
MCD
Membranous
MPGN
Mesangio Proliferative
Histopath Type
Stage1
Stage2

PREVALANCE OF NEPHROTIC RANGE OF PROTEINURIA

HISTOPATH TYPE	**SUBNEPHROTIC RANGE**(<3.5gms/dl)	**NEPHROTIC RANGE**	**TOTAL**
AMYLOIDOSIS	**1**	**0**	**1**
FSGS	**5**	**6**	**11**
IgAN	**3**	**5**	**8**
LUPUS NEPHRITIS	**2**	**4**	**6**
MCD	**2**	**6**	**8**
MEMBRANOUS NEPHROPATHY	**1**	**5**	**6**
MPGN	**1**	**2**	**3**
MESANGIO PROLIFERATIVE	**3**	**4**	**7**
TOTAL	**18**	**32**	**50**

The amount of proteinuria was quantified and the patients were divided in toe groups as follows

1. Nephrotic range (>3.5 gms/24 hours) – 32 (64%) patients

2. Subnephrotic range (1 – 3.4 gms / dl) – 18 (36%) patients

INCIDENCE OF ANEMIA

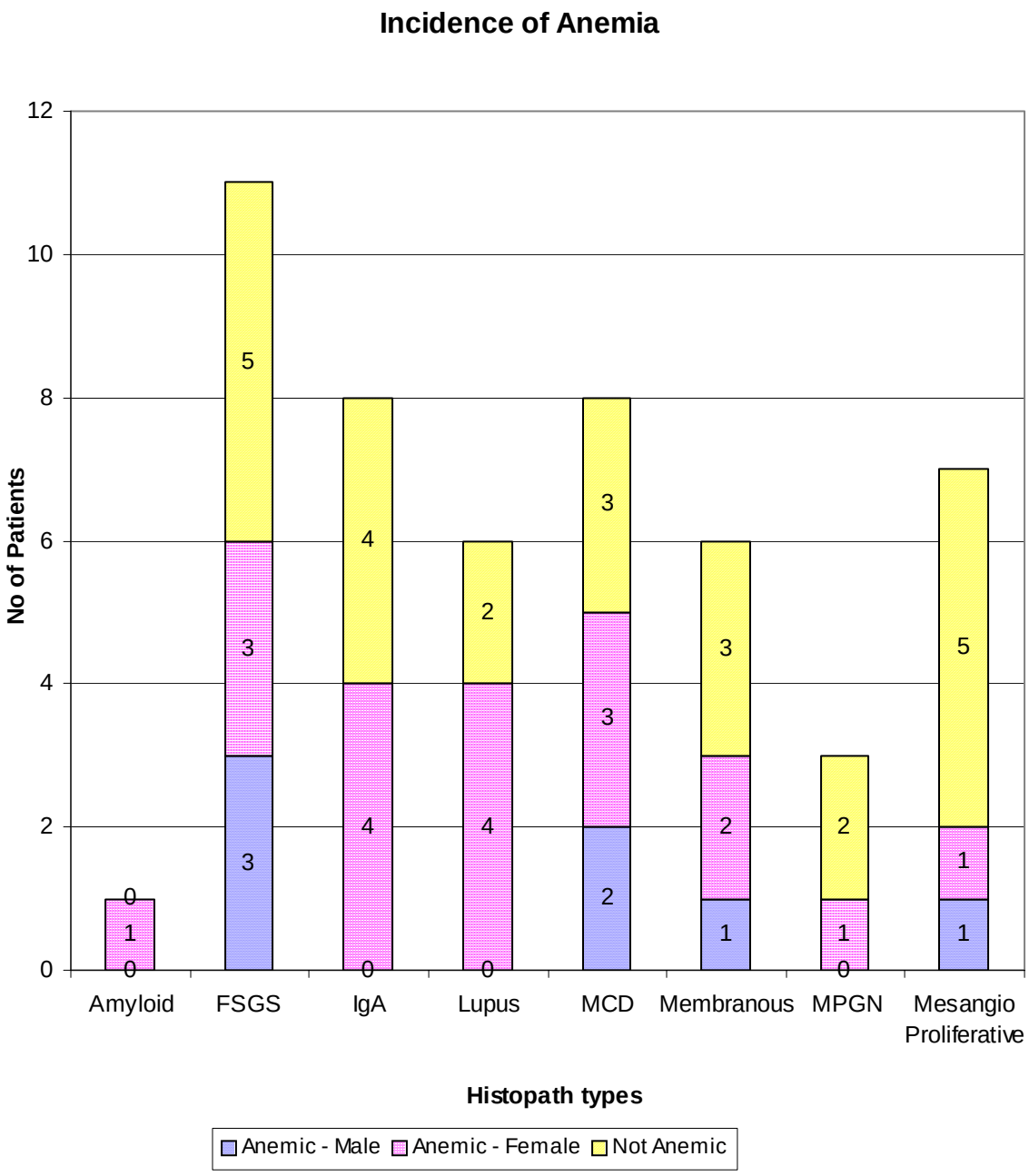
Histopathological type	Patients with Anemia (Hb <10 gms %)		
	Males	Females	Total
FSGS	3	3	6
MCD	2	3	5
Lupus nephritis	0	4	4
IgAN	0	4	4
Membranous GN	1	2	3
MesangioproliferativeGN	1	1	2
MPGN	0	1	1
Amyloidosis	0	1	1

Patients with anemia – 26

Males – 7

Females – 19

Anemia was found in 6 patients with FSGS, 5 with MCD, 4 with Lupus nephritis and IgAN each; 3 with membranous nephropathy, 2 with mesangioproliferative glomerulonephritis; and 1 each with MPGN and amyloidosis had anemia.


Incidence of Anemia
No of Patients
12
10
8
6
4
2
0
Amyloid
FSGS
IgA
Lupus
MCD
Membranous
MPGN
Mesangio Proliferative
0
1
0
3
3
5
4
0
4
4
0
2
2
3
3
1
2
3
1
0
2
1
1
5
Histopath types
Anemic - Male
Anemic - Female
Not Anemic

INCIDENCE OF HYPERLIPIDEMIA

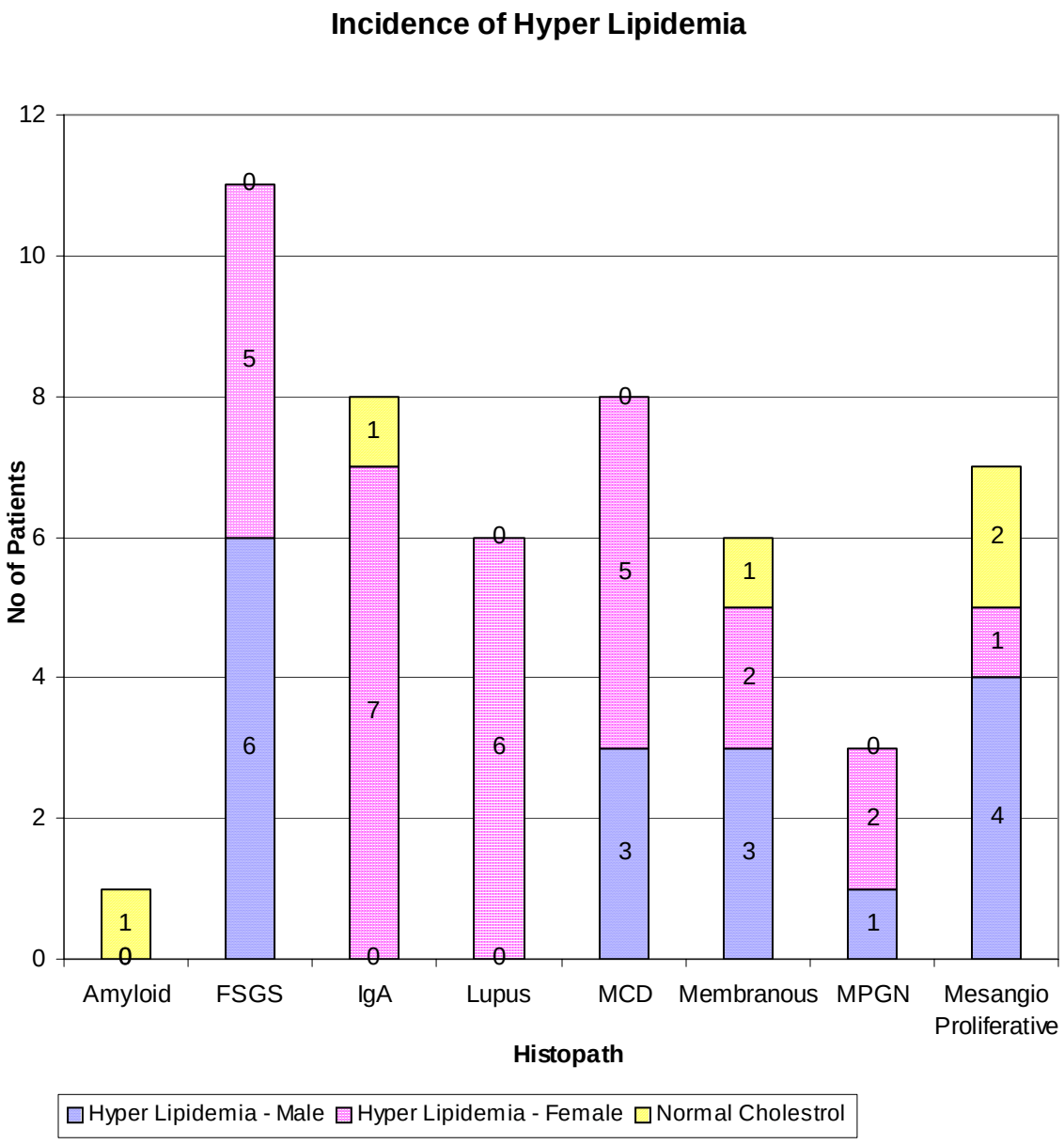
Histopathological type	Patients with Hyperlipidemia (serum cholesterol >200 mg / dl)		
	Males	Females	Total
FSGS	6	5	11
MCD	3	5	8
IgAN	0	7	7
Lupus nephritis	0	6	6
MesangioproliferativeGN	4	1	5
Membranous GN	3	2	5
MPGN	1	2	3
Amyloidosis	0	0	0

Total number of patients with Hyperlipidemia – 45

Males with Hyperlipidemia – 17

Females with Hyperlipidemia – 28

Hyperlipidemia is present in all the patients with FSGS, MCD, Lupus nephritis, MPGN, 7 with IgAN, 5 with mesangioproliferative glomerulonephritis and membranous nephropathy each.


Incidence of Hyper Lipidemia
No of Patients
12
10
8
6
4
2
0
Amyloid
FSGS
IgA
Lupus
MCD
Membranous
MPGN
Mesangio Proliferative
Histopath
Hyper Lipidemia - Male
Hyper Lipidemia - Female
Normal Cholestrol
1
0
6
5
0
7
1
0
6
0
3
5
0
3
2
1
1
2
0
4
1
2

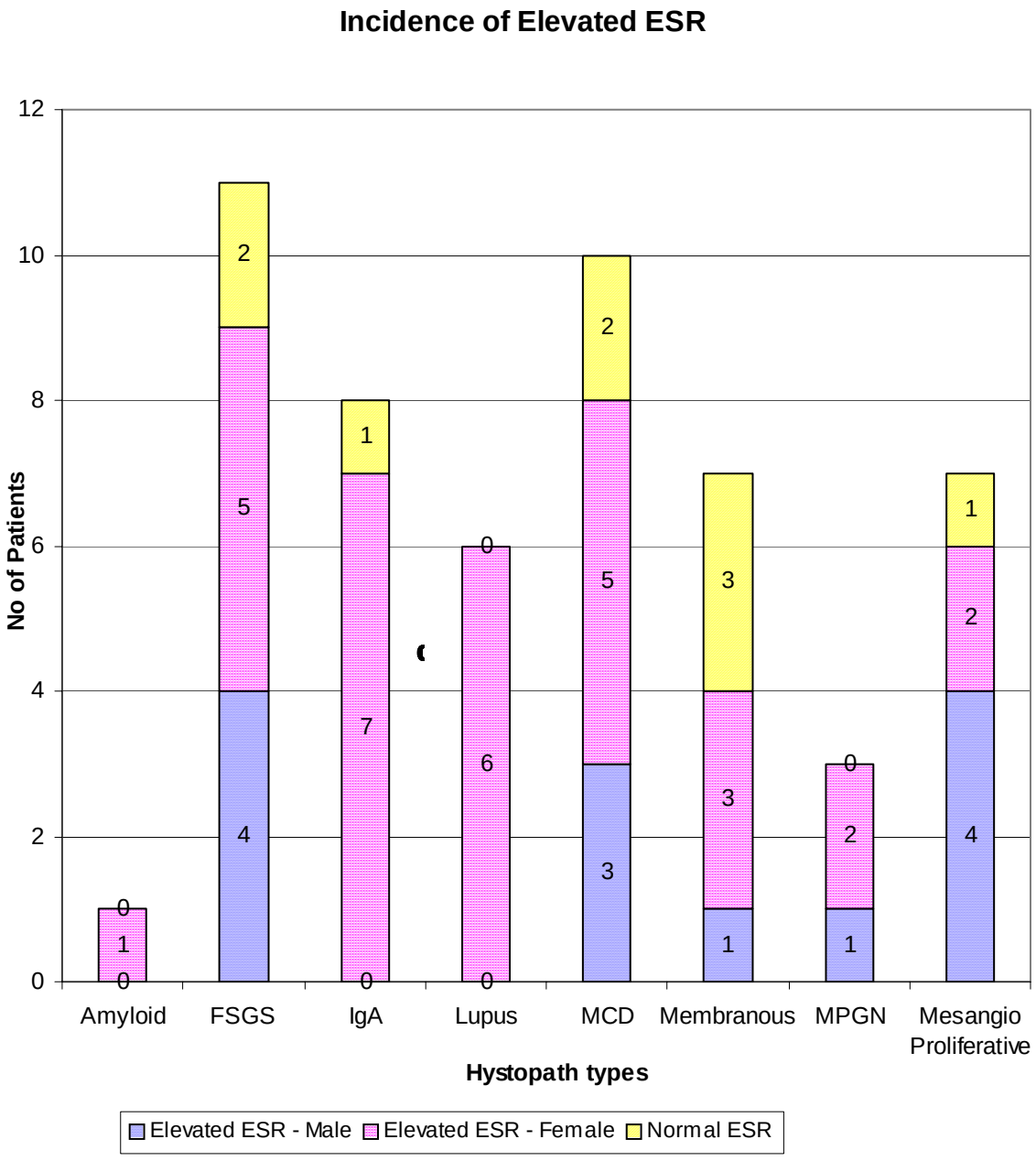
INCIDENCE OF ELEVATED ESR

Histopathological type	Patients with elevated ESR (>25mm/Hour)		
	Males	Females	Total
FSGS	4	5	9
MCD	3	5	8
IgAN	0	7	7
MesangioproliferativeGN	4	2	6
Lupus nephritis	0	6	6
Membranous GN	1	3	4
MPGN	1	2	3
Amyloidosis	0	1	1

Patients with elevated ESR – 44

Males -13

Females – 31


Incidence of Elevated ESR
No of Patients
12
10
8
6
4
2
0
2
5
4
1
7
0
0
6
0
2
5
3
3
3
1
0
2
1
1
4
Amyloid
FSGS
IgA
Lupus
MCD
Membranous
MPGN
Mesangio Proliferative
Hystopath types
Elevated ESR - Male
Elevated ESR - Female
Normal ESR

DISCUSSION

DISCUSSION

All the fifty patients included in the study were in the age group of twelve to sixty years with a mean age of 27.54 years. All the fifty patients were divided in to three groups based on the age

I – 12 to 20 years of age

There were 14 (28%) patients in this group with a mean age of 14.85 years

II – 21 to 40 years of age

There were 30(60%) patients in this group with a mean age of 30.6 years

III – 41 to 60 years of age

There were 6 (12%) patients in this group with a mean age of 44.5 years

None of the patients were above 60 years of age

Of the 50 patients included in the study 32(64%) were females and 18(36%) were males. The histopathological examination of the kidney biopsy revealed eight histopathological types. Focal segmental glomerulosclerosis (FSGS) was the commonest type found in 11(22%) patients followed by IgA nephropathy (IgAN) and minimal change disease (MCD) in 8(16%)patients each .Mesangioproliferative glomerulonephritis was found in 7(14%) patients , membranous nephropathy and lupus nephritis was found in 6(12%) patients each,3(6%) patients had membranoproliferative glomerulonephritis (MPGN) and 1(2%) had amyloidosis.

The comparison of the frequency of the various histopathological types in the present study with that of the studies conducted in various centers in India

Histopath type	**Vellore** *5* **(%)**	**Jaipur** 6 **(%)**	**Chandigarh** 7 **(%)**	**Present Study (%)**
Amyloidosis	**1.0**	**1.9**	**9.2**	**2.0**
FSGS	**16.8**	**5.3**	**29**	**22.0**
Membranous nephropathy	**9.5**	**17.4**	**4.0**	**12.0**
Mesangioproliferative GN	**7.3**	**6.7**	**10.1**	**14.0**
MPGN	**2.9**	**13.4**	**17.6**	**6.0**
IgAN	**8.4**	**7.8**	**2.0**	**16.0**
Lupus nephritis	**6.9**	**1.4**	**5.74**	**12.0**
MCD	**10.8**	**18.1**	**33.1**	**16.0**

Except FSGS and mesangioproliferative glomerulonephritis which are common in males all other types are common in females in our study which is in contrast to the study conducted in Vellore where males dominated in all histopathological types barring lupus nephritis .MCD was the commonest histopathological type in the age group of less than twenty years of age, FSGS is the commonest type in 21 to 40 years of age group and mesangioproliferative glomerulonephritis above 40 years of age. Where as in the study in Vellore MCD

was the commonest histopathological type in the age group of less than 15 years and FSGS, the commonest type in all other age groups

FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)

Of the total fifty patients studied 11(22%) had FSGS on histopathological examination and this happened to be the commonest type found. Of the 11 patients with FSGS hypertension was found in only 2(18.18%) [Against 13.3% in the study by Jayakumar et al in madras medical college, chennai] [8], renal insufficiency in 3(27.27%) [23.3% in the study by Jayakumar et al.] [8].4(36.36%) [Against 46.67% in the study by Jayakumar et al] [8]. Of the patients with FSGS had microscopic hematuria .Of the 11` patients with FSGS nephrotic range proteinuria was present in 6(54.54%) and subnephrotic range of proteinuria in 5(45.45%) patients .of the 11 patients with FSGS 2(18.18%) were in the age group of 12 to 20 years , 8(72.72%) in the age group of 21 to 40 years and 1(9.09%) above 40 years of age .none of the patients with FSGS had hypocalcaemia (serum calcium < 8gms/ dl) or hyperlipidemia(serum cholesterol >200 mg /dl) but 2(18.18%) patients had elevated ESR (>25 mm @ 1 hour)

MINIMAL CHANGE DISEASE (MCD)

Minimal change disease was found in 8(16%) of the patients studied.Of the 8 patients with MCD 5(62.50%) were in the age group of 12 to 20 years

and3(37.50%) in the age group of 21 to 40 years and no MCD was found in patients above 40 years of age. None of the patients with MCD had renal insufficiency and none of them had microscopic hematuria .6(75%) of patients with MCD had proteinuria of nephritic range 2(25%) patients had stage 1 hypertension and the remaining 6(75%) patients with MCD were normotensive anemia was found in 5(62.50%) of patients with MCD, elevated ESR in 8(100%) and hyperlipidemia in all 8(100%) patients.None of the patients with MCD had hypocalcemia

IgA NEPHROPATHY

Of the 50 patients studied 8 (16%) patients had IgA nephropathy.All the 8 patients with IgAN were females.Among the eight patients with IgAN 3(37.5%) patients were in the age group of 12 to 20 years , 5(62.50%) in 21 to 40 years age group and none were above 40 years of age .In patients with IgAN 7(87.50%) patients had renal insufficiency[68.7% in study of A Bakshi et al] [9] .Of the 8 patients with IgAN 6(75%) had hypertension[53.1 in the study of A Bakshi et al] [9] . 2(25%) patients with stage 1 and 4(50%) with stage 2 hypertension . Microscopic hematuria was found in 7 (87.50%) patients [81.2% in the study of A Bakshi et al] [9]. Nephrotic range of proteinuria was found in 5(62.50%) of patients with IgAN and 3(37.50%) had non-nephrotic range of proteinuria. Anemia was found in 4(50%) of patients with IgAN. Elevated ESR in 7(87.50%),

hyperlipidemia in 7 (87.50%) and hypocalcemia in 3(37.50%) were found in patients with IgAN.

MESANGIOPROLIFERATIVE GLOMERULONEPHRITIS

Of the 50 patients studied 7(14%) had mesangioproliferative GN, 5(71.42%) of the patients were male [64.92% males in the study of Usha et al] [10] and the remaining 2(28.57%) were females. Mesangioproliferative GN was common in the age group of 21 to 40 years with 4(57.14%) of patients in that age group, 1(14.28%) was below 20 years, and 2(28.57%) were above 40 years of age. Renal insufficiency was found in 4(57.14%) of the total 7 patients with mesangioproliferativeGN [18.33% in the study of Usha et al] [10]. 4(57.14%) patients had stage 1 hypertension, 2(28.57%) had microscopic hematuria [5.78 in the study of Usha et al] [10].4(57.14%) patients had nephrotic range proteinuria and 3(42.85%) patients had subnephrotic range. Anemia was found in 2(28.57%) patients, elevated ESR in 6(85.71%), hyperlipidemia in 5(71.42%).

MEMBRANOUS NEPHROPATHY

6(12%) of the 50 patients studied had membranous nephropathy. This type was more prevalent in the age group of 21 to 40 years, 4(66.66%) patients were in this age group and 1(16.66%) patients were in the age group of less than 20 years and more than 40 years each. Renal failure was prevalent in only 1(16.66%)

patient with membranous nephropathy [< 10% in the study of Noel LH et al 32% in the study of AD Parekh et al] [11] and Hypertension was prevalent in 3(50%) of the 6 patients with membranous nephropathy [40% in the study of AD Parekh et al] [11] and all the 3 had stage 2 hypertension. Hematuria was prevalent in 2(33.33%) patients [30-50% in the study of Noel LH et al [16] and 44% in the study of AD Parekh et al] [11]. Nephrotic range of proteinuria was prevalent in 5(83.33%) of patients with membranous nephropathy and only 1(16.66%) had subnephrotic range of proteinuria. Anemia and elevated ESR was found in 3(50%) of patients with membranous nephropathy and hyperlipidemia was prevalent in 5(83.33%) of the patients.

LUPUS NEPHRITIS

Of the 50 patients studied 6(12%) of them had lupus nephritis and all the 6(100%) were female.4 (66.66%) of the 6 patients with lupus nephritis were in the age group between 21 to 40 years and 1(16.66%) each in the other two age groups. Hypertension and renal insufficiency were prevalent in 4(66.66%) with lupus nephritis [83% Hypertension and 72% renal insufficiency in the study of A Bakshi] [9]. 4(66.66%) had nephrotic range proteinuria [against 25% in the study of Wallace et al] [12] and 2(33.33%) patients had subnephrotic range. 3(50%) patients with lupus nephritis had microscopic hematuria .All the 6(100%) with lupus nephritis had hyperlipidemia and elevated ESR 4 (66.66%) had anemia.

MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS (MPGN)

3(6%) had MPGN among the 50 patients studied. 2(66.66%) of the patients were in the age group of 21 to 40 years and 1(33.33%) patient was below 20 years of age .All the 3(100%) patients with MPGN had renal insufficiency and microscopic hematuria was found in 2(66.66%) patients, 2(66.66%) of patients with MPGN had proteinuria in the nephrotic range and 1(33.33%) in subnephrotic range. All the 3(100%) patients with MPGN had hyperlipidemia and elevated ESR 1(33.33%) patient had anemia.

AMYLOIDOSIS

Only one among the 50 patients studied had amyloidosis. The patient with amyloidosis had renal insufficiency, microscopic hematuria, subnephrotic range of proteinuria and normal blood pressure. Anemia was present in the patient,ESR was elevated no hyperlipidemia was present .

RENAL INSUFFICIENCY

Among the various parameters taken for the study renal insufficiency was found in 23(46%) patients, against 47.16% in the study conducted in Visakhapatnam [13], 45% in the study in Bangalore [14]. Among the 23 patients with renal insufficiency 17 were females and 6 were males. None of the 8(100%) with MCD had renal insufficiency, whereas all the 3(100%) patients with MPGN,

1(100%) patient with amyloidosis, 7 (87.5%) of patients with IgAN[68.7% in the study of A Bakshi et al.] [9], 3 (27.27%) of 11 patients with FSGS [23.3% in the study of A Bakshi et al] [9], 4(66.6%) of 6 patients with lupus nephritis[72% in the study of A Bakshi et al.] [9], 1(16.6%) of 6 patients with membranous nephropathy [<10% in the study of Noel LH et al[16] and 32% in the study of AD Parekh et al] [11], 4(57.14%) of 7 patients with mesangioproliferative GN [18.33% in the study of Usha et al.] [10], had renal insufficiency. All the patients with renal insufficiency were divided into three groups depending on the serum creatinine value as follows

I – Serum creatinine 1.5 to 2.9 mgs/dl

II – Serum creatinine 3.0 to 4.9 mgs/dl

III – Serum creatinine > 5.0 mgs / dl

Among the 23 patients with renal insufficiency 14 patients fell in-group I, 6 in-group II, 3 in-group III. 15 (50%) of the 30 patients in the age group of 21 to 40 years, 3(50%) of the 6 patients in the age group above 40 years of age had renal insufficiency. whereas only 5(35.71%) of the 14 patients in the younger age group of 12 to 20 years had renal insufficiency.

MICROSCOPIC HEMATURIA

On urine microscopic examination >3RBCs / HPF were taken as positive for hematuria. Among the total 50 patients studied 20(40%) had microscopic hematuria, against 37.8% in Chandigarh [7], 77.35% in Vishakapatnam [13] and 41.3%

in Lucknow study [15]. Among the 20 patients with microscopic hematuria 13 were females and 7 were males. According to the age group 7(50%) of the patients in the age group of 12to 20 years, 11(36.66%) in the age group of 21 to 40 years and 2(33.33%) in the age group above 40 years had microscopic hematuria. None of the patients with MCD had hematuria, whereas 7(87.5%) of 8 patients with IgAN had microscopic hematuria [81.2% in the study of A Bakshi et al.] [9], similarly 2 (66.66%) of the 3 patients with MPGN, 3(50%) of 6 patients with lupus nephritis, 4(36.36%) of 11 patients with FSGS [46.67% in the study of Jayakumar et al.] [8], 2(33.33%) of 6 patients with membranous nephropathy [against 30-50% in the study of Neol LH et al [16] and 44% in the study of AC Parekh et al. [11]], 2 (28.57%) of 7 patients with mesangioproliferative nephritis had microscopic hematuria [30% in the study of Usha et al] [10].

HYPERTENSION

Of the 50 patients studied 22(44%) had hypertension (>140/90 mm Hg), against 33.2% in Chandigarh and 25.9% in Lucknow studies. Of the 22 patients with hypertension 7(31.81%) were male and 15(68.18%) were female. According to the age group only 2(9.09%) were in the age group of 12 to 20 years, 17(77.27%) in the age group of 21 to 40 years and 3(13.63%) were in the age group of 40 to 60 years had hypertension. The prevalence of hypertension was highest in patients with IgAN where 6(75%) of 8 patients had hypertension

[53.1% in the study of A Bakshi et al.] [9] .The prevalence of hypertension was least in patients with FSGS where only 2(18.18%) of 11 patients had hypertension 13.3% in the study of Jayakumar et al] [8]. 4(6.66%) of 6 patients with lupus nephritis [83% in the study of A Bakshi et al], 2(25%) of patients with MCD [against 20-30% in Nolasco F, et al. – study] [17], 3(50%) of patients with membranous nephropathy [40% in the study of AD Parekh et al.] [11] , 4(57.14%) of patients with mesangioproliferative GN and 1(33.33%) of 3 patients with MPGN had hypertension[against 30%] .Of the 22 patients with hypertension 13(59.09%) had stage 1 (BP 140-159/90-99 mm Hg) and 9(40.90%) patients had stage 2 (BP > 150/100 mm Hg) hypertension.

PROTEINURIA

In all the patients studied 24-hour urine sample was collected, the amount of proteinuria was quantified and patients were divided into two groups as follows. Patients with nephrotic range of proteinuria (>3.5 gms/ 24 hours) and Subnephrotic range of proteinuria (1 –3.4 gms / 24 hours) .32 (64%) of the fifty patients had nephrotic range and 18 (36%) had subnephrotic range of proteinuria .The average amount of proteinuria in 24 hours collection was 6.29 grams. The mean amount of proteinuria was highest in patients with MCD (7.17 gms / 24 hours) [5.13 gms in the study of A Bakshi et al] [9], followed closely by patients

with FSGS (6.13 gms/ 24 hours) [4-6 gms in the Southwest Pediatric Nephrology Study group [18] and 3.31 gms in the study of Jayakumar et al. [8]] and membranous nephropathy (5.3 gms/ 24 hours) [2.58 gms in the study of A Bakshi et al.[9]]. Nephrotic range of proteinuria was present in 6(54.54%%) of patients with FSGS, 5(62.5%) of patients with IgAN, 4(66.66%) of patients with lupus nephritis [against 25% in the study of Wallace et al [14]], 6(75%) of patients with MCD , 5(83.33%) of patients with membranous nephropathy , 2(66.6%) of patients with MPGN [against 80% in the study of Mallik NP et al] and 4(57.14%) of patients with mesangioproliferative GN .

SUMMARY

1. 50 patients with proteinuria more than 1 gram per 24 hours above the age of 12 years were biopsied for renal histopathological examination. There were 32(65%) females and 18(36%) males, with a mean age of 27.54 years.

2. The commonest histopathological type found was FSGS in 11(22%) patients followed by, IgAN in8 (16%), MCD in 8(16%), mesangioproliferative in 7(14%), membranous nephropathy in 6(12%), lupus nephritis in 6(12%), MPGN in 3(6%) and amyloidosis in 1(2%).

3. MCD was commonest histopathological type in age group of less than 20 years of age, FSGS in 21 to 40 years and mesangiproliferative glomerulonephritis above 40 years of age group.

4. 23(46%) of the 50 patients had renal insufficiency and is common in patients with MPGN (100%), amyloidosis (100%), IgAN (87.5%) and lupus nephritis (66.66%). None of the patients with MCD had renal insufficiency.

5.20(40%) of the total patients studied had microscopic hematuria

6. 22(44%) of the 50 patients studied had hypertension. The prevalence of hypertension was highest in patients with IgAN.

7. 32(64%) of patients had nephrotic range of proteinuria; where the mean amount of proteinuria was 6.29 grams per 24 hours. The prevalence of nephrotic range of proteinuria was highest in patients with MCD, where 5(83.33%) had nephrotic range of proteinuria.

CONCLUSION

- The most common histopathological type was Focal segmental glomerulosclerosis (FSGS).
- Renal insufficiency was common in patients with Membranoproliferative glomerulonephritis (MPGN).
- Hypertension was common in patients with IgA nephropathy (IgAN).
- Nephrotic range proteinuria was common in patients with Minimal change disease (MCD).
- Microscopic hematuria was common in patients with IgA nephropathy (IgAN).

REFERENCES

1.Hugh R Brady, Yvonne m o'meara, Barry m Brenner.Glomerular Diseases.In: Dennis L Kasper, Eugene Braunwald, Anthony S Fanci, Dan L Longo, Stephen L Hauser, J Larry Jamsen editors. Harrison's Principles of Internal Medicine, 16th edn: McGraw Hill; 2005:1674- 1693.

2. John Savil and A J Roes.Mechanisms of Glomerular Injury. In: Oxford Textbook of Clinical Nephrology, 2nd edn:Oxford University Press:Vol II: 403-438.

3. J stewart Cameron. The Patient With Proteinuria and/or Haematuria. In: Oxford Textbook of Clinical Nephrology, 2nd edn: Oxford University Press: Vol II: 441-459.

4. J Bernheim , Z koretc. The Utility and Interpretation of Renal Biopsy. In: Anil K Mandal, editor. Textbook of Nephrology, 1st edn: Jayapee brothers; 1993:183-197.

5. Spectrum of biopsy proven renal disease and changing trends at a tropical tertiary care centre 1990 – 2001. N Blakrishnan, GT John, A Korula, J Visalakshi, GS Talulikar, PP Thomas, CK Jacob.Department of Nephrology, Pathology and

Biostatistics, Christian Medical College, Vellore, Tamil Nadu. Indian Journal of Nephrology, 2003; 12(5).

6. Profile of Renal Histopathology from a Tertiary Hospital in Rajasthan. Hareesha Babu K, Sharma LC, Agarwal D, Pathak R. Institute of Renal Sciences, SMSMC and Hospital, Jaipur. Indian Journal of Nephrology, 2005; 15(3).

7. Adolescent Onset Nephrotic Syndrome in India:Clinical featyres and Histopathological spectrum. N Sajith, K Sud, H S Kohil, KL Gupta, K Joshi, V Sakhuja. Department of Nephrology and Pathology, Postgraduate Institute of Medical Education and Research, Chandigarh. Indian Journal of Nephrology, 2001; 1(3).

8. Idiopathic Adult FSGS: Outcome of Long Term Steroid Therapy and Clinicopathological Correlation. P Nagarajan, S Kandasamy, T Muthukumar, M Edwin Fernando, R Manorajan, R Venkatraman, S Sreedhar, M Jayakumar. Department of Nephrology, Madras Medical College and Government General Hospital, Chennai. Indian Journal of Nephrology,2004;14(3).

9. Clinicopathological Spectrum of IgA Nephropathy – A 5 Year Study. A Bakshi, R W Minz, K Joshi, V Sakuja. Indian Journal of Nephrology, 2003; 14(8).

10.Clinico Payhological Features of Mesangio Proliferative Glomerulonephritis. Usha, Singh RG, Prakash J, Chaturvedi V, Kimar P, Murthy AS. Department of Immunopathology and Nephrology, IMS, BHU, Varnasi. Indian Journal of Nephrology, 2005;15(3).

11.Membranous Nephropathy- Our Experince. AD Parekh, PA Thaker, JK Pandiya, JM Vakil, NH Shah, PP Shah, HL Trevedi.IKDRC, Ahmedabad .Indian Journal Of Nephrology, 2004;14(3).

12. Clinical and pathological characteristics of patients with glomerular diseases at a university teaching hospital: 5 year prospective review, KW Chan, TM Chan, IKP Cheng. HKMJ, 1999;5(3).

13. Murphy BF, Fairley KF, Kincacid- Smith PS. Idiopatic membranous glomerulonephritis: Long-term follow-up in 139 cases.Clin Nephrol 1998; 30:175.

14. Wallace DJ, et al. Lupus nephritis.Experience with 230 patients in a private practice from 1950-1980. Am J Med 1982; 72:209.

15. Clinicopathological spectrum of childhood nephrotic syndrome.J Kumar, S Gulati, RK Sharma,A gupta, A Sharma U Gupta and RK Gupta, Departments of

Nephrology and Pathology, SGPGIMS, Lucknow. Indian Journal of Nephrology, 2001; 1(3).

16. Noel LH, et al. Long-term prognosis of idiopathic membranous glomerulonephritis: study of 116 untreated patients. Am J Med 1979; 66:82.

17. Nolasco F, et al. Adult onset minimal change nephrotic syndrome: a long – term follow-up.Kidney Int 1986; 29:1215.

18. South west Pediatric Nephrology Study Group. Focal segmental glomerulosis in children. A report of the southwest Pediatric Nephrology Study Group. Kidney Int 1985; 27:442.

19. Nephrotic Syndrome – Analysis of 1720 Renal Biopsies. Suresh Babu V., Girish Narayen, Anuradha, Swarnalatha G. Department of Nephrology, Osmania General Hospital and Department of Pathology, Apollo Hospital, Hyderabad. Indian Journal of Nephrology, 2001; 1(3).

20. Analysis of 490 Kidney Biopsies: Data from The United Arab Emirates Renal Disease Registry. Journal of Nephrology, 1998; 11(3):148-150.

21. Membranoproliferative glomerulonephritis is still the most frequent glomerulonephritis in Lithuania. L.Razukevicience, I.A.Bumblyte, V.Kuzminskis and A.Laurinavicius. Clinical Nephrology, 2006; 65(2):87-90.

22. Increasing incidence of focal segmental glomerulosclerosis and an examination of demographic patterns.Clinical Nephrology, 2005; 63(1):1-7.

23. Date, A., Raghavan, R., John, T.J., Richard, J., Kirubakaran, M.G., and Shastry, J.C.M. (1987).Renal disease in adult Indians: a clinicopathological study of 2827 patients. Quartely Journal of Medicine, 1987; 64: 729-37.

24. Sud A, Bhuyan UN, Tandon HD. Spectrum of Morphologic Lesions in Renal Biopsies Associated With Primary Nephrotic Syndrome. Ind J Med Res 1978; 68: 811.

25. Renal biopsy analysis of patients with nephrotic syndrome in a tertiary hospital.Kavitha S.,Shobhana G.Nayak, Urmila.,Surjit S., Loyd V. Dept.of nephrology, St.John's Medical College Hospital, Banglore. Indian Journal Of Nephrology ,2001;1(3).

26. Audit of renal biopsies at Trivandrum Medical College from July 1 2001 – June 30 2004.B Upendran, S Uthup, MK Mohandas, KP Karunan,JGeorge, A Vimala.Medical Colleg , Trivandrum, Kerala. Abstracts, 35th Annual Conference of Indian society of Nephrology, Indian Journal of Nephrology, 2004; 14(3).

PROFORMA

PROFORMA

NAME:

AGE:

SEX:

N.C no:

24-HOUR URINARY PROTEIN (Gms/dl):

URINE RBC'S:

BLOOD UREA:

SERUM CREATININE:

HEMOGLOBIN:

TOTAL LEUKOCYTE COUNT:

ESR:

SERUM CHOLESTEROL:

SERUM ELECTROLYTES:

BLOOD PRESSURE:

ULTRASONOGRAM OF KIDNEY

- **RIGHT KIDNEY SIZE:**
- **LEFT KIDNEY SIZE:**
- **CORTICAL ECHOGENECITY:**

RENAL BIOPSY - HISTOPATHOLOGY:

MASTER CHART

S.no	Name	Age	Sex	Proteinuria gms/24hr	Urine RBC	Urea	Creatinine	Hb gms	TC	ESR	S.Cholestrol	S.Protein A/G	Na^{+}	K^{+}	ca^{++}	BP	RK	LK	Echo	Histopathology
1	Suganya	13	F	3.6	0	38	1.2	7.4	9000	20/40	296	3.5/2	136	4.4	9.8	110/90	12.8	12.2	G1	MCD
2	Priya	16	F	2	2	62	1.9	7.4	9900	68/132	228	3.6/2.1	135	4.1	10.1	130/80	10.6	10.5	G0	MPGN

3	Lalitha	25	F	2.8	3	82	3.2	10.6	8600	2/8	284	3/2	136	1/3	8.9	140/90	9.3	9.7	G2	IgAN
4	Jeeva	31	F	3.6	0	60	1.7	9	9000	40/72	352	3.7/1	138	4.3	9.4	130/70	10.1	10.2	G1	FSGS
5	Ameen	35	F	5.4	0	20	1	7	8600	80/168	216	3.6/1	138	4	10	140/90	9.2	9.6	G1	LUPUS
6	RathinaKumari	34	F	7.2	20	118	4.6	9.4	9200	35/76	273	3.6/1	138	4.4	9.6	160/110	9.1	9.5	G1	IgAN
7	Boopalan	30	M	8.1	0	22	1	11.2	7400	5/12	272	3.5/2.1	128	4	9.5	130/80	9.4	9.6	G2	Mes PGN
8	Lakshmi	20	F	7.28	0	20	1	9.2	8600	20/4	323	3.6/2.2	138	3.9	9.8	110/70	11.1	10.9	G0	Memb PGN
9	Valankanni	30	F	4.8	0	20	1	10.2	10000	1000/68	262	3.3/2	138	4	9.9	110/70	9.5	9.7	G1	FSGS

10	Sa…	35	F	5.4	0	48	1.2	8.8	6900	50/105	312	3/2	136	3.7	8.9	120/70	10.8	10.7	C0	MCD
11	Sureka	14	F	12	0	35	1	10.6	8200	45/70	560	1.9/2	136	3.7	10	100/70	12	11.8	C0	MCD
12	Ashok	16	M	12	0	16	0.8	10.2	6200	2/5	226	3.6/2	138	4	9.5	120/60	9.6	9.8	C1	MembPGN
13	Krishnaraj	36	M	12	0	30	1.3	11	7200	5/20	301	3.2/2.1	138	4.7	9.3	130/80	10.3	9.6	C2	MembPGN
14	Varalakshmi	35	F	11.4	15	88	3	11	7900	15/140	246	3.7/2	138	4.4	7.9	180/110	9.8	10.1	C2	IgAN
15	Joel	17	F	2.1	3	20	1	11	8200	30/62	254	3.6/2	138	4	8.2	110/80	11.3	11.1	C1	IgAN
16	Kalyani	37	F	4.3	0	20	1	11	4000	94/140	229	3.5/2.1	138	3.9	8.4	140/90	10	9.9	C1	MCD

17	Manikandan	15	M	3.48	5	18	1	9.8	6900	65/135	388	3.4/2	138	3.9	8.6	130/80	9.9	9.7	G3	FSGS
18	Ravi	36	M	3	12	20	1	10.6	8300	115/15	268	3.8/2.1	138	4	9.2	160/90	9.9	10.4	G1	FSGS
19	Rukmani	40	F	3	0	90	3.1	5.8	9200	95/130	179	3.7/2	136	4.4	7.9	130/70	10.8	11	G3	AMYLOID
20	Samundeshwari	24	F	1.94	20	49	18	8.7	9530	40/85	256	3.5/2.1	129	4	9	140/90	9.7	9.9	G2	LUPUS
21	Stella	27	F	6	0	48	18	5.4	5800	65/110	324	3.6/2.1	139	4.4	8.9	120/80	9.8	10	G3	LUPUS
22	Deepa	22	F	4.8	0	78	22	10.8	8000	25/55	228	3.7/2.2	139	4.4	7.8	140/90	9.9	10.1	G1	IgAN
23	Bagavathy Murugan	37	M	2.3	0	138	56	10.6	7800	55/100	286	3.6/2	130	4.9	9.4	140/90	10.4	10.8	G1	Mes PGN

24	Manikandan	13	M	2.4	0	18	1	10.2	8400	30/64	234	3.5/2.1	136	3.7	9.6	120/80	10	10.2	G2	FSGS
25	Hemalatha	14	F	4.2	25	78	1.9	11	9600	20/42	216	3.4/2.2	138	4	9.5	130/80	9.8	10.1	G0	LUPUS
26	Moulali	35	M	12	3	18	1	10.8	9000	22/45	292	3/2	135	4.5	9.9	140/80	11.4	10.3	G0	Mes PGN
27	Aravindraj	21	M	8	2	178	7.3	8.2	7200	20/48	220	3.2/2	135	4.9	9.3	150/100	10.1	10.3	G2	Memb PGN
28	Harikrishnan	13	F	10.8	20	68	3.2	11	8200	25/60	148	3.3/2.1	138	3.9	7.9	110/70	9.8	10.4	G0	Mes PGN
29	Radha	36	F	5.5	3	68	2	9.3	9300	35/72	192	3.7/2.1	138	4.2	8	160/100	10.2	10.4	G2	IgAN

30	Samrajyam	45	F	2.52	0	26	1.1	10.6	9000	40/80	304	3.7/2.1	138	4	9	150/90	10.7	10.6	C0	Mes PGN
31	Raja	28	M	2.2	12	22	0.8	7.3	7600	20/422	242	3.6/2.1	133	3.6	9.6	110/80	10.5	9.4	C0	FSGS
32	Rajalakshmi	24	F	15	0	86	3.5	10.6	8600	54/100	311	3.3/2.2	138	3.5	8.6	130/70	10.3	10.5	C2	FSGS
33	Guna	30	F	12.5	0	30	1.2	9	10800	60/122	200	3.7/2.2	136	3.8	9.5	110/70	10.4	10.6	C1	MCD
34	Rose	26	F	2.4	0	22	1	8.2	8200	95/120	202	3.7/2.1	138	4	9.4	120/80	10.5	10.7	C1	FSGS
35	Chitra	33	F	4.8	2	26	1.1	10.8	7000	78/100	196	3.5/2.2	138	3.9	9.2	140/100	9.4	10.1	C0	Membr PGN
36	Sakthivel	23	M	9	0	50	1.6	11	8200	42/85	240	3.7/2.3	138	3.9	8.1	140/90	9.9	9.6	C0	MPGN

37	Vasu	40	M	13.9	2	62	17	9.7	9600	10/22	222	3.6/2.2	138	4.4	9.2	120/70	10.8	10.1	C1	FSGS
38	Suresh	32	M	2.4	0	72	1.9	10	9200	36/70	302	3.5/2	138	4.4	9.8	140/90	9.6	9.8	C2	Mes PGN
39	Sathya	18	F	2.6	5	140	58	5.2	1010 0	200/42	320	3.5/2.1	136	4.9	8.3	150/100	9.5	9.7	C3	IgAN
40	Aravinth	13	M	2.4	0	20	1	9	1020 0	100/33	286	3.4/2	138	4	9.4	110/70	9.5	10.2	C1	MCD
41	Avinash	13	M	2.4	0	20	1	9	1020 0	100/33	286	3/2	138	4	8.7	110/70	9.5	10.2	C1	MCD
42	Selvi	34	F	9.6	0	40	11.6	11.2	1010 0	105/110	321	3.1/2	135	4.6	9.8	140/90	11.1	10.8	C1	LUPUS

43	Savithri	49	F	2.2	8	21	0.9	8.4	4300	60/134	225	3.2/2.1	128	3	9.8	150/100	13.6	12.8	C0	LUPUS
44	Manoharan	17	M	19.6	0	18	0.8	10.8	9400	64/120	290	3.3/2.1	134	4.2	8.9	130/80	9.2	9.5	C1	MCD
45	Murugan	26	M	14.4	0	22	1	11	9000	5/14	264	3.7/1.1	138	4	10.1	140/90	11.1	10.6	C1	FSGS
46	Nagarathinam	45	F	6.8	0	84	3.4	7.8	5600	40/80	198	3.4/2.1	140	4.3	10	120/80	10.6	10.8	C2	Mes PGN
47	Logeswari	16	F	10.4	6	76	2.3	9.3	7800	38/80	257	3.5/2.2	136	4	9.3	130/80	10.7	10.9	C1	IgAN
48	Thangam	48	F	1.9	0	54	1.4	8.4	8300	64/120	312	3.6/2.2	135	3.9	8.3	160/90	10.9	11.3	C0	MembPGN
49	Muthamizh	24	F	3.7	2	62	1.7	10.4	9600	48/100	307	3.7/2.2	132	4.1	9.7	130/80	10.8	11.1	C2	MPGN

50	Srikala	36	F	2.3	0	36	1.3	9.8	7200	30/60	224	3.4/2	136	4	9.4	120/70	9.9	10.1	C1	FSGS